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A.M.A. ARCHIVES OF OTOLARYNGOLOGY

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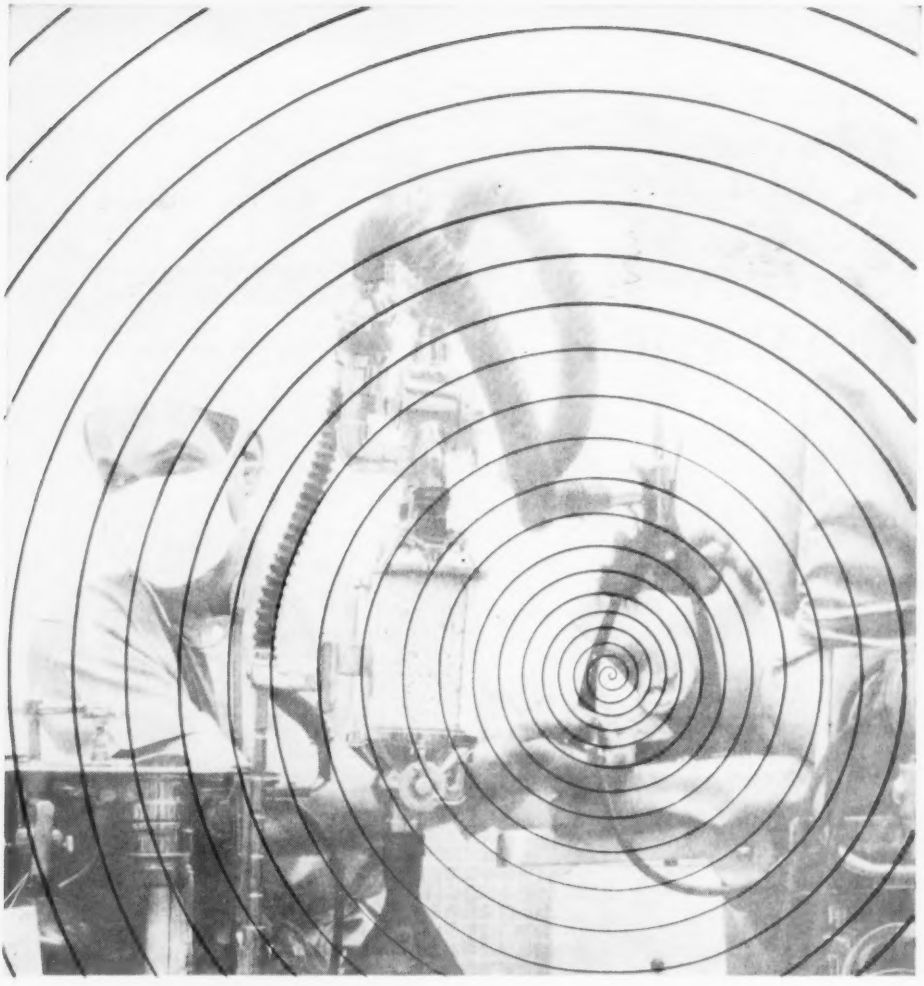

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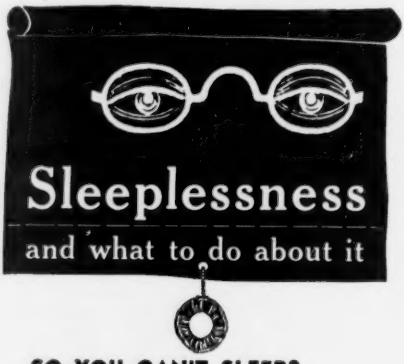

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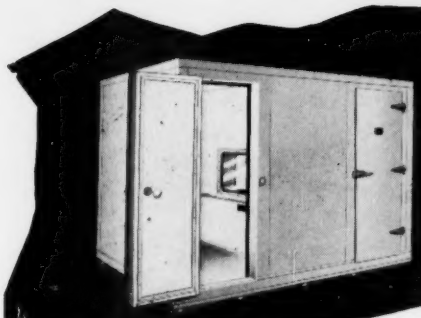

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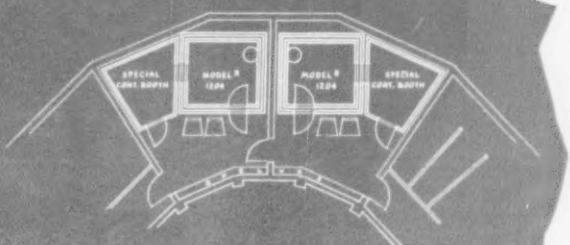

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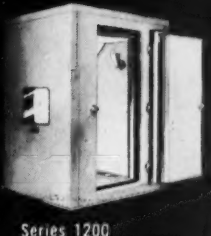

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The Problem of Idiopathic Bleeding Time Defect in Laryngology

ROBERT L. GOODALE, M.D., Boston

The purpose of this paper is to call to the attention of laryngologists the condition of "idiopathic bleeding time defect" which Jacobson, working at the Massachusetts General Hospital, has described. In this paper I wish to give all credit to Dr. Jacobson for the hematological study of the disease and for his aid in its control. As I am a laryngologist and not a hematologist, I would like to quote from Jacobson's report on 100 cases,[1] the following paragraphs in order to make clear the precise characteristics of idiopathic bleeding time defect.

"A number of these patients might be classified as persons with pseudohemophilia." In a footnote Jacobson objects to this term, as the condition in the patient with idiopathic bleeding time defect "does not resemble hemophilia in either the hereditary aspects, the sex distribution, or in the type of bleeding." "In brief, patients with this syndrome include persons of either sex who have a proclivity to bleeding spontaneously or after trauma in the skin, from the mucous membranes. . . . The characteristic laboratory findings . . . include a normal number of circulating platelets; a normal or slightly prolonged coagulation time, with normal retraction of the clot; a normal or abnormal degree of capillary fragility as determined by the tourniquet test, but a bleeding time that is usually significantly prolonged."

Jacobson states further that the pathogenesis is still uncertain but "in all probability represents a defect in capillary permeability with probably little or no participation in the defect by humoral factors." His patients ranged in age from 3 months to 80 years. About 65% were female. The sites of bleeding were quite unlike those of hemophilia. Spontaneous bleeding in the skin, either marked ease of bruising or purpuric eruptions, was noted in about 40% of the cases. Frequent epistaxes occurred in 50%. In females over 12 years of age, half gave a history of menometrorrhagia. Bleeding from the gastrointestinal tract, kidney, and central nervous system was very frequent. In five persons, the premonitory episode involved the retina and vitreous. Bleeding, of course, followed all types of trauma, but, unlike that seen in hemophilia, trauma to joints and muscles was never followed by bleeding. One quarter of the patients gave a history of excessive bleeding following dental extractions; another quarter, after tonsillectomy, and the trauma of rhinoplasty was followed by severe bleeding in five cases.

In Jacobson's series the tendency toward remission during pregnancy was noted in 10 patients. In regard to treatment he notes that administration of blood in any form had no influence on the bleeding or bleeding

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time nor had there been any improvement from ascorbic acid, vitamin P (rutin, hesperidin), calcium, vitamin K or the newer carbazochrome (adrenochrome [semicarbazone], commercially called Adrenosem). Good results, however, have followed the use of corticosteroids in doses far in excess of those used in rheumatoid arthritis and bronchial asthma. Jacobson's determination of bleeding time is quite different from the use of needle prick, in which there is no uniformity in size of needle or depth of penetration.[2]

A more accurate method is to elevate the forearm to the same level as the heart with the patient either reclining or sitting. An area on the volar aspect of the forearm, free of visible superficial veins is sterilized by the application of a sponge soaked in acetone, with avoidance of rubbing of the skin. A new, previously unsterilized No. 11 Bard-Parker blade is soaked for a few minutes in acetone. A 3 mm. ink mark is made on the sterilized portion of the forearm and similarly a transverse ink mark is made on the blade 3 mm. from the tip after the acetone has evaporated. A sphygmomanometer cuff around the upper arm is inflated to 40 mm. Hg and kept at this level throughout the test. The blade is thrust into the skin to a depth of 3 mm. and drawn distally the length of 3 mm. parallel to the 3 mm. ink mark. The arm is tilted so that the blood flows away from the incision and is packed up by filter paper at intervals of 30 seconds, without the paper touching the incision. After the bleeding has ceased, the edges of the wound are brought together by a "bandaid" type of dressing.

Ordinarily the bleeding does not begin significantly until about one minute later and is bright red in color. The upper limit of normal appears to be 6½ minutes; the range being 3½ and 5½ in the vast majority of cases.

One condition which may obscure the findings in this test is free aortic regurgitation, where the high pulse pressure is too great for the normal hemostatic factors. But this condition can be readily detected prior to the test.

As laryngologists we are all subject to emergency calls from our medical confreres to stop a persistent epistaxis or to control what seems to be intractable postoperative hemorrhage. I would state that all procedures which are now generally practiced, such as packing, cauterizing, tying or suturing, hospitalization for sedation and transfusion, and, of course, for adequate laboratory study, are absolutely necessary.

I do not at all intimate in this paper that corticosteroids are the only agents to consider. But idiopathic bleeding time defect can frustrate all these commonly accepted procedures, and from personal experience I strongly urge its consideration. To illustrate, I will give the following brief notes from my own cases.

Case 1.—A married woman, 51 years old, with a history of metrorrhagia until menopause three years previously had always had a tendency toward epistaxis. Blood pressure unstable, suddenly rises when excited but usually is 130 systolic. When first seen in consultation, on July 14, 1951, the patient had had epistaxis off and on for four weeks, increasing in severity, uncontrolled by packing and cauterization. She was transferred to the Massachusetts Eye and Ear Infirmary. Blood studies showed R. B. C. 2,150,000, Hgb 8.1, platelets increased. She had a transfusion and anterior and postnasal packing. The patient had two more hemorrhages on the following two days. Dr. Jacobson, in consultation, found all blood tests normal with exception of a low red cell count and Hgb and a bleeding time of 16½ minutes. The patient was given cortisone, 100 mg. at eight-hour intervals. Bleeding time fell to three minutes by the end of the first week. The cortisone was reduced during the following three days to 50 mg. every eight hours, but the bleeding time increased again; so a maintenance dose of 75 mg. was determined upon, which kept her bleeding time at 6½ minutes. There was no further bleeding.

Case 2.—An unmarried woman, 30 years old, was seen in consultation because of an upper respiratory infection complicated by repeated attacks of acute secretory otitis media. She also complained of almost constant earaches. She had had many attacks of tonsillitis. Nasal examination was negative; sinuses were negative; tonsils were chronically infected, and an adenoid was definitely enlarged. Eustachian tubes were easily infected. Both drums were slightly dull, with questionable retraction. Hearing test showed combined conduction and perceptive loss for both ears of 30 to 40 db. In view of the definite evidence of tonsil infection, a tonsilladenoidectomy was advised. However, as in addition to the above history she had metrorrhagia, it was decided to have the patient enter the hospital for preoperative blood studies. In consultation with Dr. Jacobson, there was discovered an idiopathic bleeding time defect

of 15½ minutes. The blood was otherwise negative. Preoperatively, she was given cortisone 300 mg. and a salt-free diet daily for four weeks. She did not gain weight and showed no drug complications. Bleeding time was 8½ minutes on the day of operation. Tonsillectomy was done. By this time the adenoid had subsided, and, in view of a still prolonged bleeding time, it was not removed. During the operation there was enough bleeding to necessitate suturing, but no other bleeding problem was encountered from then on. On Dr. Jacobson's recommendation, the cortisone was reduced to 50 mg. t. i. d. during the next seven days and then omitted. However, she was advised to resume it during her next menstrual period.

CASE 3.—A married woman, 21 years old, was seen on Jan. 3, 1956, because of repeated attacks of tonsillitis with accompanying cervical adenitis. She also had had a history of spontaneous purpuric bleeding in the legs for the past year and one episode of severe epistaxis and metrorrhagia. A tonsilladenoidectomy was advised. Preoperative bleeding time by the usual needle technique was one minute. This was checked by Jacobson's method and found to be 11 minutes 30 seconds. Other blood findings were normal. The following regimen was followed: For two days preoperatively prednisone (Meticorten) 25 mg. with Gelusil 8 cc. by mouth every six hours. During the operation 100 mg. of hydrocortisone in 1,000 cc. of 5% dextrose in water at 40 drops per minute; this was repeated once again during the same postoperative day. Cortisone, 100 mg. I. M. t. i. d., for the next three days. Then prednisone, 20 mg. t. i. d., for three days. The subsequent course was uneventful. Although the bleeding time on admission was 11½ minutes, on the day of operation it was down to 7 minutes. At discharge from the hospital it was 8½ minutes, still somewhat elevated; so she was continued on prednisone for a few weeks after going home.

CASE 4.—A married woman, 23 years old, was referred by her father-in-law, a physician, for a tonsilladenoidectomy as she had had very frequent severe attacks of tonsillitis for the past years. She also was pregnant. There was a known bleeding history. Three years previously she had a severe hemorrhage following a tooth extraction. She also had metrorrhagia. Her bleeding time was checked and reported to be 9½ minutes. Blood was otherwise normal. The same regimen was followed in this case, only with smaller dosage as the prolongation was rather less than in Case 3. Preoperatively prednisone 60 mg. daily was given in divided (t. i. d.) doses. On the day of operation she received 200 mg. of hydrocortisone in 2,000 cc. of dextrose in water 40 drops a minute. Postoperatively prednisone 20 mg. t. i. d. was given for one week, then 15 mg. t. i. d. for two weeks. This patient had no further bleeding problem and went to term satisfactorily. It may be that she had relatively less of a problem because of pregnancy, as Jacobson noted in 10 other cases.

CASE 5.—A schoolgirl, 15 years old, was referred for pyocele of the right ethmoid. For six months there had been noted an increasing hard swelling in the medial wall of the right orbit. Subsequently this was incised in another city, without change in condition. So far as known there was no bleeding problem then; however, no extensive surgery was attempted then either. When first seen by me, she had a small scar just medial to the inner canthus on the right and just beneath this a hard visible swelling. From an x-ray this was reported to be a pyocele of the ethmoid. Operation was done by the external route, and only moderate bleeding was encountered. However, she bled postoperatively and then gave a history of ocasional severe purpuric bleeding. Bleeding time was then found to be prolonged to 10½ minutes. The patient was put on prednisone. After discharge from the hospital, she was followed subsequently by Dr. Jacobson, who reported that after she discontinued medication one week after going home, she had two minor epistaxes, and that one month postoperatively she still had a bleeding time of 14½ minutes. I saw her one month later. She had no further bleeding.

The above five cases illustrate several problems which can bother us and raise the question of standing preoperative procedures. I spoke of this at a panel discussion at the American Academy of Otolaryngology and Ophthalmology a year ago, and some one in the class asked whether I advocated routine preoperative blood studies on all cases. I do not think that the question is just a matter of ordering studies. We should be able to narrow down our laboratory work by a careful inquiry into the bleeding problem, just as we try to eliminate unnecessary x-rays and other tests by a good history taking. Let us use our intelligence more and make use of the laboratory only when the history and physical examinations are unable to supply essential knowledge.

If the history is at all suggestive of a bleeding problem, I most certainly would order blood studies several days in advance of an operation in order to start medication in time to reduce the bleeding time. In such a case, it is my feeling that it would be

much wiser to have a standing order for certain studies as a precautionary screening. I would specify blood tests as follows: R. B. C., Hgb, W. B. C. and differential, platelets, prothrombin, clotting time, and bleeding time according to Jacobson's technique.

Conclusions

1. There are a number of people who have a normal blood picture in every respect except that their bleeding time is significantly prolonged.

2. The needle or stylet method which is generally used and which shows a normal of three minutes is not adequate. The scalpel incision as described by Jacobson is more accurate and will reveal the true bleeding time in cases pronounced normal by the older method.

3. A regimen for the control of idiopathic bleeding time defect is available.

4. More careful screening preoperatively of suspected bleeders is recommended.

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Effect of Filtration on the Chemical and Irritating Properties of Cigarette Smoke

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Introduction

Cigarette smoke is an aerosol consisting of a suspension of minute particles in a vapor and gaseous phase. Many factors affect its composition. These include the composition of the tobacco, physical characteristics of the cigarette, and technique of smoking, among others. In addition to the above parameters, there are two principles involved in cigarette smoke modification by filtration: "mechanical filtration," in which there is simple retention of the particulate (solid) matter, and "adsorption filtration," which consists of selective removal of certain vapors or gases due to the unique properties of some materials of high surface area such as activated charcoal. The tobacco itself in a cigarette acts as a mechanical filter.

It might not be amiss to indicate at this point the confusion which exists, especially with reference to the particulate (solid) content of cigarette smoke inasmuch as this is the fraction that contains the nicotine and so-called "tar" to which so much attention has recently been directed. For instance, one laboratory reported values on "tars" which were 66% higher than those found by another laboratory in tests made at the same time and on the same brand of cigarettes, despite the high degree of uniformity in established brands of cigarettes. Lack of standardization in terms of the aforementioned factors capable of affecting the composition of cigarette smoke undoubtedly plays a role in accounting for discrepancies of this type. In addition, not a little misunderstanding is created by the absence of agreement as to the definition of the term "tar" itself. Some apply this term to the total particulate (solid) phase of cigarette smoke, while others apply it to that portion of the particulate phase which is soluble in certain solvents such as chloroform and toluence.[1,2]

Despite the growing popularity of filter-tip cigarettes, there is relatively little information on the effect filters have on cigarette smoke other than a reduction in solids due to mechanical filtration. Apparently, little attention has been given to the potentialities of adsorption filtration.

About 10 years ago we reported in this journal[3] on the efficacy of certain filter tips in reducing cigarette smoke irritation, concluding that the three tips studied were no more effective in filtration than the column of tobacco they replaced. The filters were of the mechanical type. During the interim there have appeared several papers dealing with the chemical aspects of filter tip efficiency. The most extensive of these studies were those reported from the Chemical Laboratory of the American Medical Association[4,5,6] and by Keith and Newsome.[2] Here again the filters under observation were of the mechanical type. To our knowledge, no systematic biological studies have been reported in this field since the appearance of our paper referred to above.

Recently reports have been presented describing the quantitative separation of the particulate phase[1] and the gaseous and vapor phase[7] of cigarette smoke. These methods make it possible to determine the individual roles of mechanical and adsorp-

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TABLE 1.—*Effect of Filtration on the Particulate Constituents of Cigarette Smoke (Average Values from Six Determinations on Ten Cigarettes Each)*

Cigarette	Total Solids		Chloroform-Soluble Fraction		Nicotine	
	Mg/Cigt.±S. D.*	% Reduction	Mg/Cigt.±S. D.	% Reduction	Mg/Cigt.±S. D.	% Reduction
All tobacco	31.4±0.42	—	13.8±0.23	—	1.77±0.028	—
Plain composite filter tip	25.1±0.50	20	11.3±0.40	18	1.48±0.04	16
Impregnated composite filter tip	23.2±0.36	26	10.3±0.19	25	1.44±0.031	20

* = Standard deviation.

tion filtration in evaluating the over-all efficacy of filter tips from a chemical point of view. It is the purpose of the present paper to present data demonstrating the effect of these two principles of filtration on the chemical composition and the concomitant irritating properties of cigarette smoke.

Procedures

The same mixture of blended tobacco was used in making up three lots of 85 mm. (king size) cigarettes. Lot 1 (all-tobacco) had no added filter; Lot 2 (plain composite filter tip) had 15 mm. of the tobacco column replaced by a filter consisting of 50% cellulose acetate plus 50% soft paper (mechanical filter); Lot 3 (impregnated composite filter tip) had a similar column of tobacco replaced by a filter consisting of 50% cellulose acetate plus 50% soft paper impregnated with activated charcoal (mechanical plus adsorption filter). For purposes of standardization cigarettes selected for study weighed within ± 20 mg. of the average weight of the lot and contained approximately 12% moisture.

Chemical analysis of the smoke for materials in the particulate phase was done by the method of Wartman and Harlow,[1] in which the particulate matter is trapped on a suitable glass fiber disc fitted into the filter holder. The results reported were obtained through the cooperation of the Research Laboratory of the American Tobacco Company. Determination of gaseous and vapor phase constituents was done by the method of Irby and Harlow,[7] which employs a gas chromatographic technique. The results reported are those given in their paper covering the analyses done on the same lots of cigarettes that we have studied biologically.

The irritation studies were conducted according to the procedure we used in our previous study on filters and was originally described by Finnegan et al.[8] Essentially this is as follows:

The rabbit eye is used as a convenient site for testing, since it represents a true mucous membrane and under pentobarbital anesthesia may be easily exposed to whole smoke samples without previous surgical manipulations. Of the tissues of the rabbit eye, the membranous lining of the upper lid was found to show a graded response in the form of edema to increasing smoke dosage. The amount of edema produced was measured by excising the membrane, weighing, drying, and reweighing it, and calculating the wet to dry weight ratio. The difference in the ratios between the treated and control eyes was used as an index of the edema produced.

In these experiments a modification of an automatic smoking machine described by Bradford, Harlan and Hanmer[9] was used to draw three 35 ml. puffs of smoke, spaced at one-minute intervals, across the well-opened eye of the anesthetized rabbit, 10 of which were used for each lot of cigarettes. Excision of the tissues for the previously described weighings was made one hour after ex-

TABLE 2.—*Effect of Filtration on the Gaseous and Vapor Phase Constituents of Cigarette Smoke (Average Values from Nine Determinations on Four Cigarettes Each)*

Cigarette	Constituents Mg/Cigt.*										
	Acetaldehyde	Isoprene	Acetone	Methyl Alcohol	Methyl Ethyl Ketone	Acrolein	Propionaldehyde	Isobutylaldehyde	Methyl Furan	Furan	Methyl Acetate
All tobacco	0.73	0.63	0.39	0.09	0.086	0.07	0.050	0.033	0.028	0.031	0.015
Plain composite filter tip	0.66	0.55	0.37	0.071	0.076	0.066	0.048	0.031	0.026	0.030	0.012
Impregnated composite filter tip	0.50	0.39	0.20	0.025	0.038	0.036	0.027	0.019	0.014	0.018	0.009

* Standard deviations calculated for all components varied from 2% to 8% and were for most components about 4%.

TABLE 3.—*Effect of Filtration on the Gaseous and Vapor Phase Constituents of Cigarette Smoke*

Cigarette	Constituents Percentage Reduction										
	Acetaldehyde	Isoprene	Acetone	Methyl Alcohol	Methyl Ethyl Ketone	Acrolein	Propionaldehyde	Isobutylaldehyde	Methyl Furan	Furan	Methyl Acetate
Plain composite filter tip	9.6	12.6	5.1	21.1	11.6	5.7	4.0	6.1	7.1	3.2	20
Impregnated composite filter tip	31.5	38.1	48.7	72.2	55.8	48.6	46.0	42.4	50.0	41.9	40

posure to the first puff. The identity of the filter "tipped" cigarettes remained unknown until the tests were completed.

Results

Table 1 gives the results of the chemical analysis for materials in the particulate phase in the mainstream smoke of the three lots of cigarettes. In comparing the values obtained on the smoke from the all-tobacco cigarettes with those obtained on the smoke from the plain composite filter-tip cigarettes, it is seen that the plain composite tip decreased the total solids by 20%; the chloroform-soluble fraction by 18%, and the nicotine content by 16%. Comparable figures for the impregnated composite tip are 26%; 25% and 19%.

Table 2 gives the results of the chemical analysis of certain vapor phase constituents of the mainstream smoke from the three lots of cigarettes.

In contrast to the rather modestly increased efficiency of the impregnated composite filter over the plain composite filter in removing particulate matter, the figures in Table 3 demonstrate its comparatively remarkable ability to reduce vapor phase components. For instance, while the plain composite filter caused only slight decreases in the vapor phase constituents, the impregnated composite caused marked decreases in all the compounds analyzed. The reductions resulting from the use of impregnated composite filter ranged from 31.5% to 72.2%.

Table 4 lists the results of the irritation tests made on the mainstream smoke of the three lots of cigarettes along with a statistical analysis of the differences between their irritation values. It is obvious from these data that the charcoal-impregnated filter was significantly more effective in removing edema-producing irritants from the smoke than was the plain filter which actually was no more effective than the column of tobacco for which it was substituted.

Comment

On comparing the effect of these two types of filters on the chemical composition of cigarette smoke with their effect on its

TABLE 4.—*Effect of Filtration on the Edema-Producing (Irritating) Properties of Cigarette Smoke (Average Values from Results on Ten Rabbits on Each Lot of Cigarettes)*

Cigarette	Average Ratio Difference ± S. D. *	P (Probability) Values Between Lots as Determined by Analysis of Variance †		
		All Tobacco	Plain Composite Filter	Impregnated Composite Filter
All tobacco	3.97±1.17	—	N. S.	<.01
Plain composite filter tip	3.31±1.47	N. S.‡	—	<.05, >.01
Impregnated composite filter tip	2.07±0.94	<.01	<.05, >.01	—

* S. D. = standard deviation.
† A "P" value of < .05 is considered significant and one of < .01 highly significant.
‡ N. S. = not significant.

irritating potency, one is not surprised at the findings in light of what is known concerning the irritating properties of individual cigarette smoke components. Mechanical filtration, as exemplified by the plain composite filter tip, removes primarily nicotine and "tar." These cigarette smoke constituents are not objectively acutely irritating.[10,11] The impregnation with charcoal to produce adsorption filtration, in addition to mechanical filtration, led to a significant reduction in the vapor phase constituents. Many of these vapor phase components, especially the aldehydes, have known acutely irritating effects.[12] Hence a combined filter embracing the principles of both mechanical and adsorptive filtration has this added property over filters based on mechanical principles alone.

Summary and Conclusions

Studies have been made on the chemical and irritating properties of the smoke of three lots of identical cigarettes except that one lot had no filter; one lot had a plain cellulose acetate-soft paper mechanical filter, and one lot had a composite mechanical-adsorption filter in which the soft paper component had been impregnated with charcoal as the adsorptive agent.

It was found that on chemical analysis the composite charcoal-treated filter was only very slightly more effective than the plain composite filter in removing the particulate phase components, notably nicotine and "tar," from the smoke. However, the charcoal-impregnated composite filter was many times more effective than the plain composite filter in reducing the components of the vapor phase of the smoke.

These chemical changes can be correlated with the results obtained from comparative studies made on the irritating properties of the smoke from the three lots of cigarettes as measured by the edema-producing potencies of the smoke on the conjunctiva of anesthetized rabbits. The removal of nicotine and "tar" by mechanical filtration was not reflected by a significant decrease in the irritating properties of the smoke. On the other hand, the decrease in the vapor phase constituents due to adsorptive filtration effected by addition of charcoal to the filter led to a parallel decrease in the irritating properties of the smoke. These findings are what might have been expected on the basis of the known irritating properties of individual cigarette smoke constituents.

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Histopathology of the Facial Nerve in Herpes Zoster Oticus

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Herpes zoster is a common nervous disease known since ancient times. Denny-Brown, Adams, and Fitzgerald, in an article based upon contributions of von Bärensprung, Head and Campbell, Lhermitte and Nicolas, and three cases of their own, sketched the broad outlines of this condition as follows:

"The disease is probably due to a filtrable virus, similar to, if not identical with, that of varicella, which provokes an acute inflammatory reaction in isolated spinal or cranial sensory ganglia, the posterior gray matter of the spinal cord and the adjacent leptomeninges. The clinical manifestations are a vesicular cutaneous eruption, radicular neuralgia and, less often segmental palsies and sensory loss. . . .

"In view of the large number of sensory ganglia which may be involved, the clinical possibilities are manifold. In the region of the cranial nerves two special syndromes are frequent, namely, "opthalmic herpes" and "geniculate herpes." The pathologic changes in the Gasserian ganglion in the former syndrome were described by Head and Campbell. The combination of paralysis of the facial nerve, auricular herpes, with or without deafness, tinnitus and vertigo, was first ascribed to a herpetic lesion of the geniculate ganglion by Ramsay Hunt."

Herpes zoster oticus is a benign, even if often a most painful and annoying disease, and consequently microscopic examinations of the facial nerve are extremely rare, only three cases having been reported. Consequently we feel that our findings should be published, although we realize that they are of limited value as very little material (the temporal bone only) was at our disposal for examination. The patient was examined and treated by one of us (G-M) in Ringsted Hospital.

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Otologist to Ringsted Hospital (Department of Internal Medicine: Head, J. Piper, M.D.), Dr. Guldberg-Möller; Assisting Prosector to the Department of Pathology, Municipal Hospital, Copenhagen (Head, S. Petri, M.D.), Dr. Olsen; Head, Department of Otolaryngology, Frederiksborg County Central Hospital, Dr. Kettel.

Report of a Case

Woman 78 years of age. Prior to this illness the patient's health had been fairly good. Ten years ago she had suffered from attacks of vertigo accompanied by nausea and impaired hearing on the left side. Seven years prior to admission she had been examined by an otologist, who described the drums as normal. Whispered voice was heard at a distance of 4 meters with the right ear but could not be heard on the left side. There was no spontaneous nystagmus, and the caloric reactions were normal. Bellergal * was administered with good effect.

Two days before admission she was examined by one of us (G-M) because of renewed attacks of vertigo. The drums were normal, but she could hear conversational voice only within a distance of 0.3 meter on the left side, and a spontaneous nystagmus to the right side was observed. At this time there were no signs of facial palsy. The right ear was normal.

The same evening she started complaining of pain in the left ear irradiating toward the angle of the jaw, and of an increasing swelling of the left external ear and the tongue. Within two days a complete peripheral facial palsy had developed. The temperature had risen to 39 C (102.2 F), and she continued to experience vertigo.

She complained of headache, but not of nausea or vomiting.

On admission there was a complete peripheral facial palsy on the left side. The external ear was heavily swollen and red, with an extensive vesicular eruption and crust formation on dried vesicles. The external auditory canal was completely closed by vesicles.

* A proprietary sedative containing Bellafoline (a combination of the malic acid salts of the total levoratory alkaloids of belladonna leaves), Gynergen (ergotamine tartrate), and phenobarbital.

There was no hearing on the left side, and spontaneous nystagmus towards the right side was observed.

The tongue was red, and on the left side vesicles and fibrin membranes were observed on the anterior two-thirds. Consequently it was impossible to examine the taste.

She had a feeling of dryness in her left eye, and on examination there was no lagophtalmos. Lumbar puncture was not done.

Chlortetracycline (Aureomycin) and dihydroergotamine were administered, and three weeks after admission the eruption in the left ear had disappeared.

The external auditory canal now looked normal, but the drum was somewhat sclerotic. She was completely deaf on the left side, and the facial palsy was complete. The spontaneous nystagmus, however, had disappeared.

Six weeks after admission she suddenly died from embolus of the pulmonary artery, verified at autopsy. Twenty-four hours later the temporal bone was removed.

The temporal bone, including the labyrinth, looked macroscopically normal. There was no tumor in the internal auditory meatus, and the cerebrum and meninges appeared normal. The Fallopian canal was opened without anything macroscopically abnormal being observed. The intratemporal part of the facial nerve, including the geniculate ganglion, was removed for examination.

The nerve was cut in serial sections and stained after the method of Van Gieson, with hematoxylin-eosin, and with gallocyanin, as well as for myelin sheaths, and silver-impregnated for axis cylinders.

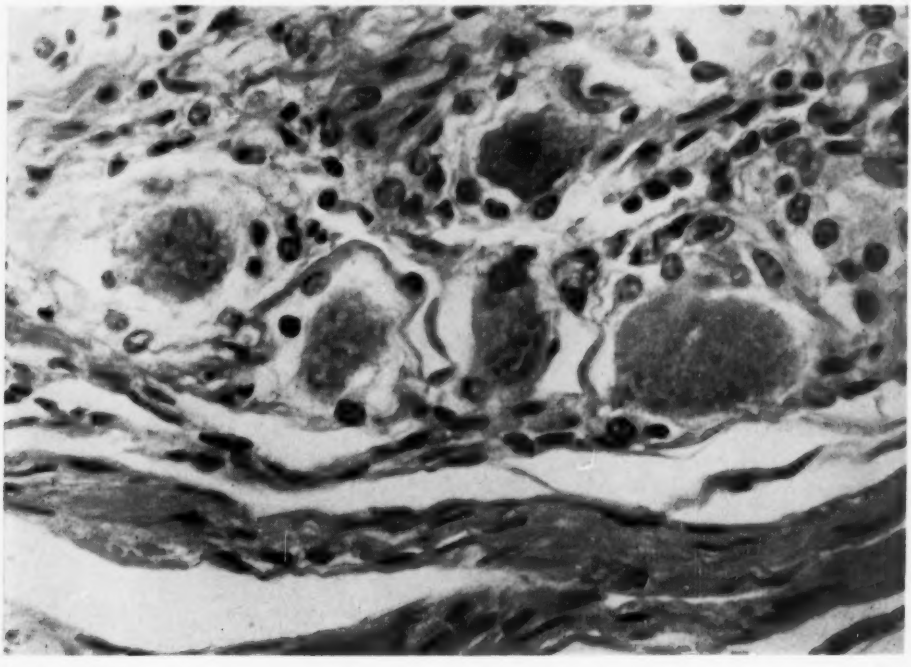
The histologic examination gave the following results:

1. *Geniculate Ganglion.*—Only slight alterations were observed. The ganglion was not swollen. A very moderate diffuse decrease of nerve cells was suspected. The majority of the cells were normal, some, however, slightly degenerated, shrunken, and pyknotic.

Only a few lymphocytes were observed, and nowhere was necrosis seen.

2. *Facial Nerve.*—Along the entire intratemporal course of the facial nerve striking changes were observed. The medullary sheaths had almost completely disappeared, the remaining few being heavily degenerated. The axis cylinders were degenerated, and between them a moderate fibrosis with heavy diffuse infiltration with lymphocytes and a few plasma cells were observed. The Schwann sheaths were present, their nuclei ap-

Fig. 1.—Geniculate ganglion from case of herpes zoster oticus. Absence of nuclei in some of the nerve cells is due to the latter being out of focus. Absence of inflammatory reaction.



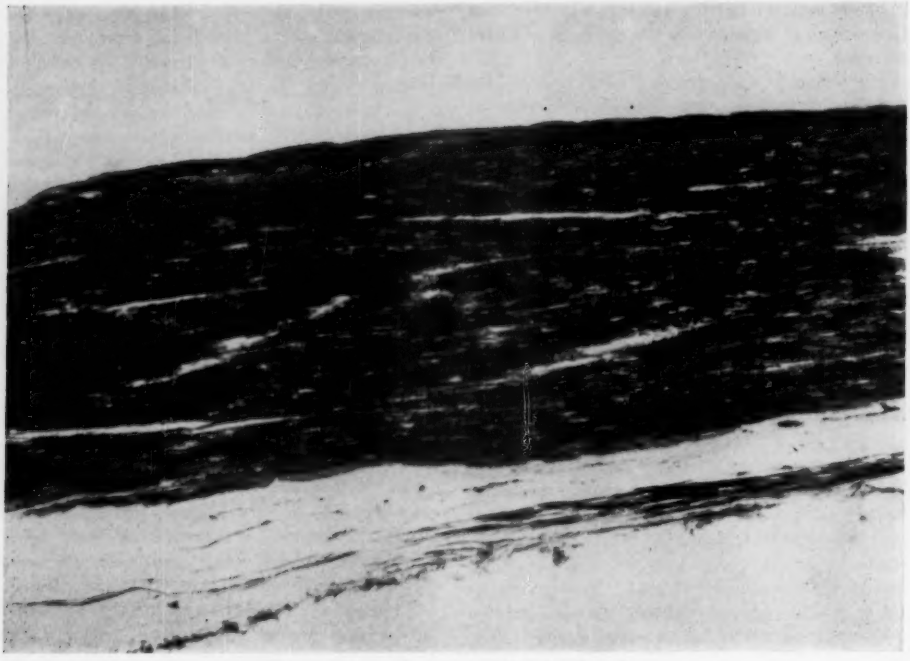
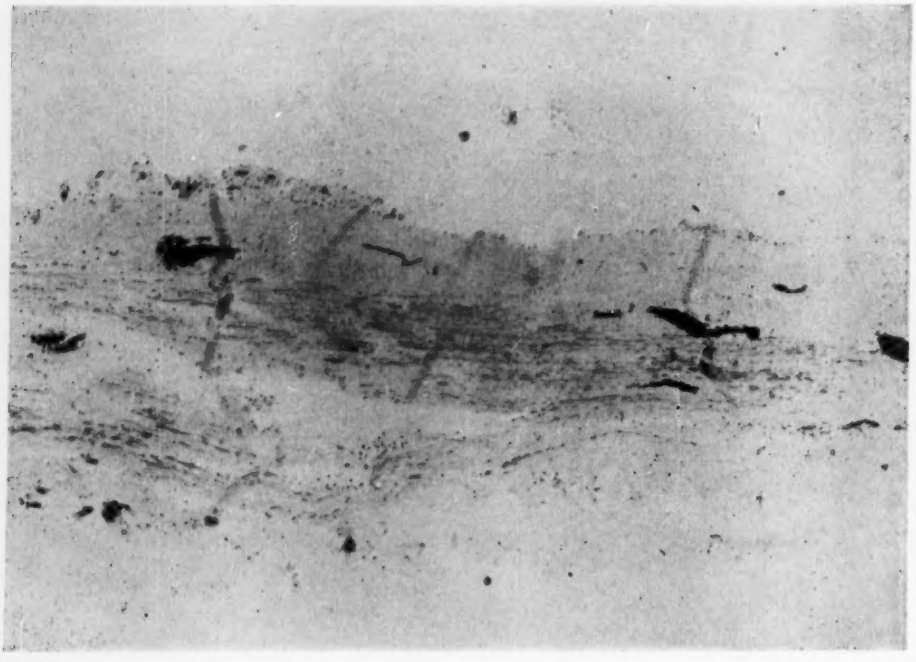


Fig. 2.—Normal nerve, myelin sheath stain.

Fig. 3.—Facial nerve from case of herpes zoster oticus, myelin sheath stain. Practically complete demyelination.



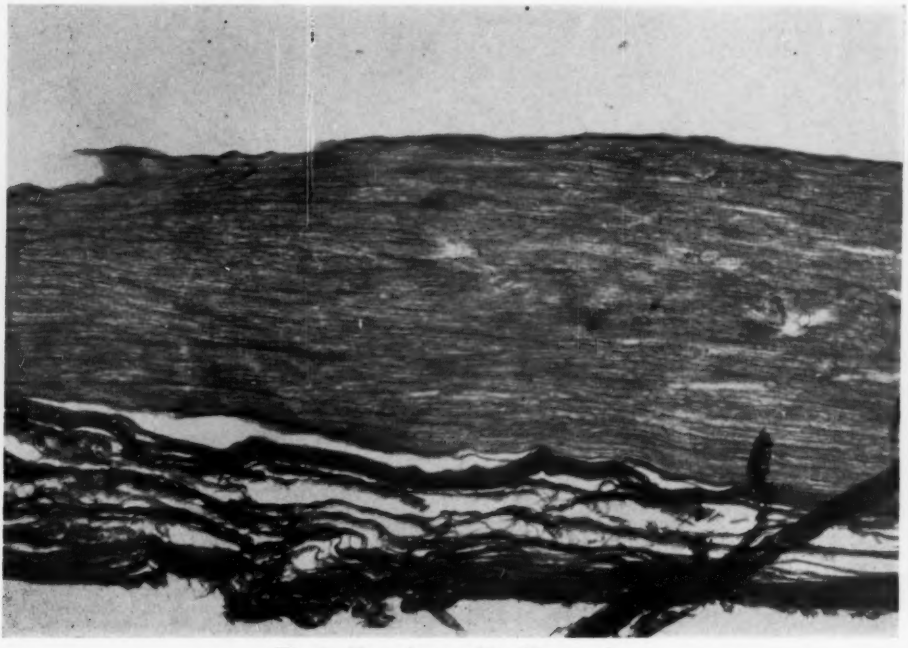


Fig. 4.—Normal nerve, Van Gieson stain.

parently proliferating. There were no signs of intraneural hemorrhage.

Summary: A woman, 78 years of age, who for six weeks prior to her death had suffered from a typical herpes zoster oticus restricted to the external meatus and cavum conchae ("geniculate zone") and the anterior two-thirds of the homolateral side of the tongue, and accompanied by complete peripheral facial palsy and aural symptoms, died suddenly from embolus of the pulmonary artery. The meninges, the brain, and the temporal bone looked macroscopically normal.

Microscopically, only slight changes were noted in the geniculate ganglion, whereas the whole intratemporal part of the facial nerve was heavily degenerated and infiltrated with lymphocytes.

Classification

Hunt postulated that a herpetic eruption in a certain area of the external ear, accompanied by pain, was due to posterior poliomyelitis of the facial nerve. In severe cases also the auditory and vestibular ganglia may be involved, either as the original site of infection or through extension from the geniculate ganglion. He proposed the following classification of the symptoms:

1. Herpes oticus (without neurologic signs)
2. Herpes oticus with facial palsy
3. Herpes oticus with facial palsy and hypoacousis
4. Herpes oticus with facial paralysis and Ménière's syndrome

Ten years prior to the herpes infection our patient had also suffered from Ménière-like symptoms. She must probably be considered as belonging to Group 4, as at the time of vesicular eruption what remained of hearing was lost and spontaneous nystagmus was present. This was probably due to the inflammatory involvement of the auditory and vestibular ganglia.

As it was impossible to examine the taste on the tongue, which was swollen and red and had vesicular eruption, we were not able to apply Tschiassny's topognosis to determine clinically the site of the lesion. It should be remembered, however, that

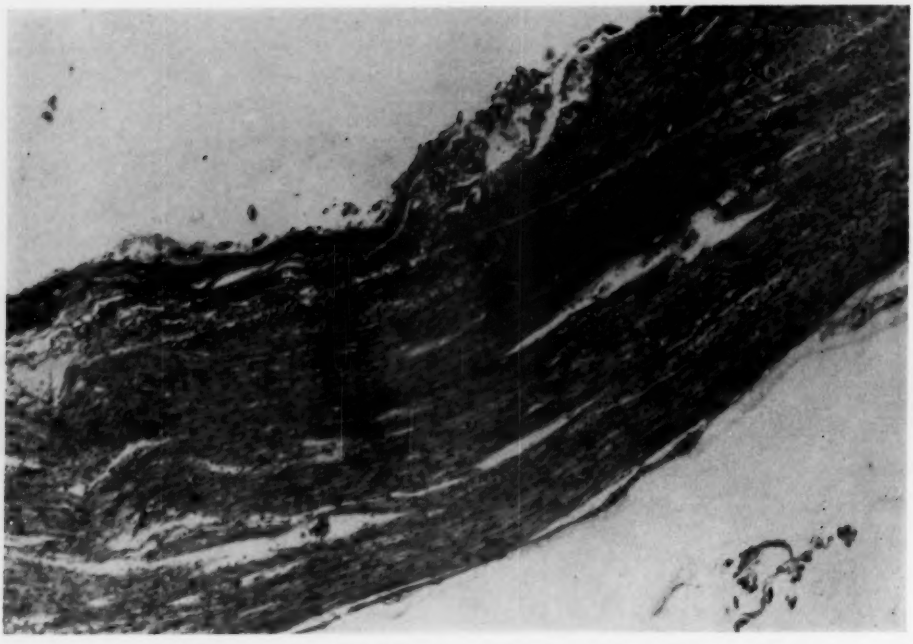
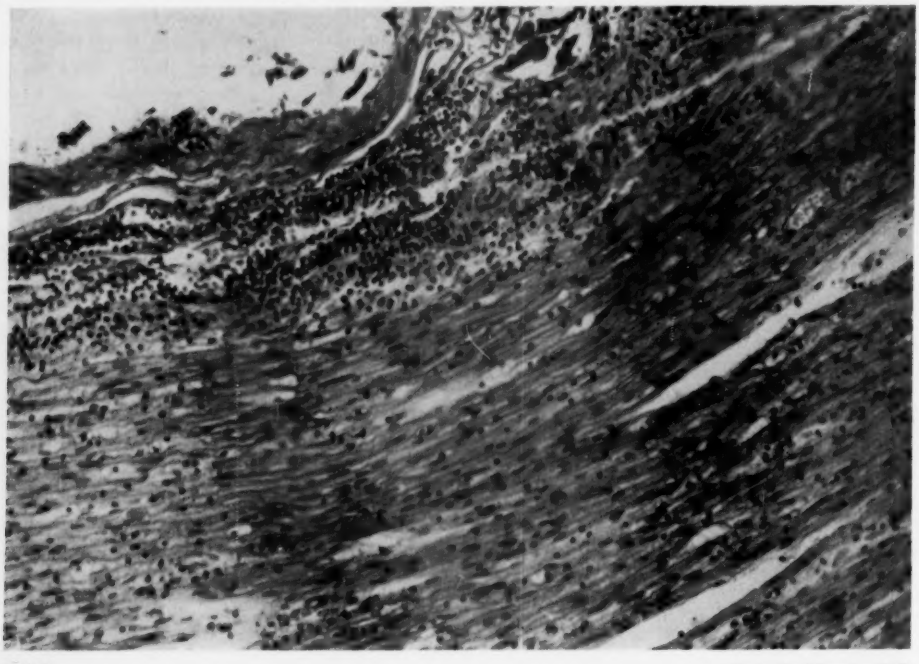


Fig. 5.—Facial nerve from case of herpes zoster oticus. Heavy lymphocytic infiltration.

Fig. 6.—Same as Figure 5 higher magnification.



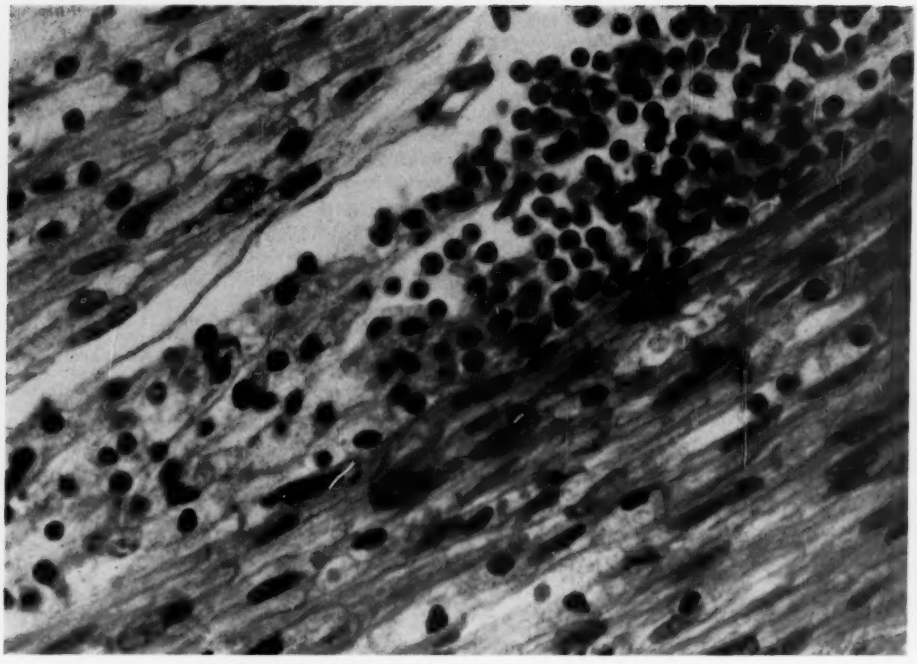


Fig. 7.—Same as Figures 5 and 6 still higher magnification.

even if a perfect examination can be completed, "topognosis cannot be regarded as a method reliable for classification when applied to the individual case" (Tschiassny).

Survey of Literature

In his many papers on the "geniculate syndrome" Ramsay Hunt described the postmortem changes in only one case, and in that the geniculate ganglion was not examined. His patient was a man 48 years of age, who died 80 days after an attack of herpes occipitocervicalis with facial paralysis. Inflammatory and degenerative changes were found in the first, second, and third cervical ganglia and corresponding posterior roots. The geniculate ganglion, unfortunately, was not examined.

Three reports including histologic examination of the geniculate ganglion were available, however, previous to the present case.

Maybaum and Druss examined a man, 51 years of age, who at the time of his death had suffered for six weeks from a herpes zoster oticus and facial palsy. Periarteritis nodosa was found in numerous organs, and also bilateral suppurative labyrinthitis and ostosclerosis.

The cells of the geniculate ganglion on the side affected were severely degenerated, shrunken, pyknotic, and disintegrated, with numerous capsule cells. The facial nerve, too, showed degeneration and was infiltrated by lymphocytes, plasma cells, and eosinophil cells. On the other side both nerve and ganglion were normal.

Findlay reported his case as follows:

The patient was a male, M. F., aged fifty-two years, with herpes zoster of the left external canal and the cavum conchæ and complete left facial paralysis, which had persisted for three months prior to his death from a ruptured gastric ulcer with peritonitis.

The result of the histologic examination is described as follows:

The left petrous temporal bone has been decalcified, and microscopic slide preparations have been made to demonstrate the geniculate ganglion and neighboring structures. Most of the nerve

cells of the geniculate ganglion are well preserved. Degenerative changes are present in a small proportion of them, some show cytoplasmic shrinkage and nuclear pyknosis, and others have lost their nuclei. There are lymphocytes and a few plasma cells scattered throughout the ganglion, plus facial nerve fibres, and there is lymphocytic cuffing of an occasional thin-walled blood vessel.

More marked inflammatory changes are present around a group of blood vessels and a nerve trunk found in the bone close to the ganglion.

Unfortunately, as in our case, only the temporal bone and not the central nervous system was examined.

A beautiful report has been published by Denny-Brown, Adams, and Fitzgerald. They described three cases of herpes zoster, in which the central nervous system was histologically examined. Case 3 was one of "auricular" and "occipito-collaris" herpes with palsy of the facial nerve:

A man 62 years of age complained of a painful rash over the head and neck, followed two or three days later by vesicular eruption in the right occipital and postauricular region. The skin in the homolateral external auditory canal was swollen, red, and desquamating.

The cervical lymph nodes were enlarged and tender on the same side. Eleven days later a complete peripheral facial palsy developed, as well as a deviation of the tongue to the right, accompanied by a new eruption of vesicles over the posterior aspect of the right side of the head. The facial palsy disappeared within three weeks. The patient died 64 days after the onset of the disease from intra-abdominal bleeding, probably caused by an ulcer of the stomach.

The pathologic and anatomic studies were extremely comprehensive, the following findings being the most significant.

1. Geniculate ganglion. The ganglion contained a normal complement of cells, none of which appeared to have suffered any damage. There was no cellular infiltration.

2. Facial nerve. Patchy infiltration with lymphocytes was observed throughout the course of the seventh nerve. In the intracranial portion of the nerve the cellular reaction was scattered and at no point intense. No certain evidence of damage to the nerve fibers was seen at this level. Immediately within the Fallopian canal there was a dense patch of lymphocytic cells, with an occasional plasma cell. Such accumulations appeared at irregular intervals throughout the remainder of the course of the nerve. One focus lay just proximal to the geniculate ganglion and another immediately distal, both clearly related to bundles of nerve fibers which did not enter the ganglion. The axis cylinders were beaded and thinned.

3. Dorsal root ganglia. Second cervical ganglion: The whole ganglion was virtually destroyed, the necrotic area surrounded by a wide zone of dense lymphocytic infiltration. Third, fourth and fifth cervical ganglia: practically normal.

4. Posterior roots in their meningeal course. The first and second severely degenerated and infiltrated with lymphocytes.

5. Spinal cord. At the level of pyramidal decussation there were perivascular and meningeal infiltrates of lymphocytes, decreasing in number at the first cervical level, while at the second cervical level large numbers of microglia cells and lymphocytes together with degeneration of some nerve cells were encountered.

The pathologic features were summarized by Denny-Brown et al. as follows: "Typical necrotizing ganglionitis (second cervical ganglion) and motor neuritis of the facial nerve without damage to the geniculate ganglion were observed."

Comments

In herpes zoster the microscopic changes in the spinal ganglia constitute the most striking and distinctive pathologic feature (Denny-Brown et al.), but extensive inflammatory changes in the central nervous system described by Lhermitte and Nicolas, Wohlwill, Faure-Beaulieu and Lhermitte, are also encountered.

In surveying the available reports and their own cases Denny-Brown et al. affirmed four histologic events which distinguish herpes zoster from other pathologic processes and which they describe as follows:

1. A ganglionitis marked by pannecrosis of all or part of the ganglion, with or without hemorrhage, and surrounded by intense lymphocytic infiltration. This phenomenon was, in all published accounts and in our own cases, associated with the eruption of vesicles characteristic of the disease in the corresponding cutaneous segment. Wohlwill alone claimed that inflammation of a nerve root or of a peripheral nerve can, without the necrotic ganglionitis, account for the zoster. 2. A poliomyelitis which closely resembles anterior polio-

myelitis but is readily distinguished by its unilaterality, segmental localization and greater involvement of the posterior horn, posterior root and dorsal spinal ganglion. 3. A relatively mild, localized leptomeningitis, in which the cellular infiltrate is relatively slight and limited principally to the involved spinal segments and nerve roots. 4. True peripheral mononeuritis, seen not only in the nerves distal to the ganglion but in the anterior nerve root, both within the meninges and in the portion contiguous to the involved spinal ganglion. These pathologic changes are substratum for the neuralgic pains, the persistent pleocytosis, and the local palsies which may attend and follow the zoster infection.

Ramsay Hunt postulated, by argument from analogy, that herpes zoster oticus was due to a geniculate ganglionitis: ". . . even in the absence of the geniculate ganglion the existence of well marked degeneration in the nerve of Wrisberg (pars intermedia of the seventh nerve) and its intrabulbar roots is sufficient proof that this structure was involved."

This statement is criticized by O'Neill: "That the geniculate ganglion may have been involved no one can gainsay, but loss of the ganglion eliminated either proof or disproof of involvement. Clinically the patient showed facial paralysis and cervical ganglionitis (verified at necropsy) and that is all. Hunt's assertion is illogical and unjustified." Maybaum and Druss's case is difficult to evaluate as the patient was suffering from a periarteritis nodosa and a bilateral suppurative labyrinthitis. Even though the alterations seem to have been more pronounced in the facial nerve than in the ganglion, even though a hemorrhagic necrosis typical of herpes zoster was not found, and even though "the lesion shown in their illustrations does not resemble that characteristic of herpes zoster" (Denny-Brown et al.), Maybaum and Druss's observation does not definitely refute Hunt's postulation. Tschiassny, however, is completely right in stating: "The simultaneous presence of a suppurative process involving the middle ear and the inner ear structures opened the door for equivocal interpretations of the histopathologic changes noticed in the geniculate ganglion."

When Findlay concerning his own case states that "most of the nerve cells of the geniculate ganglion are well preserved," and that "degenerative changes are present in a small proportion," it is hard to accept his conclusion: "The changes seen in these sections are consistent with a history of herpes of the geniculate ganglion." One must remember that an extensive herpetic eruption was found in the external canal and the cavum conchae, which are richly innervated by several other nerves. The ganglia of these nerves were not examined by Findlay, and they may have been causally related to the herpes and the site of alterations characteristic of the disease.

The patient of Denny-Brown et al. was suffering from an "occipito-collaris and auricular" herpes with palsy of the facial nerve. In this case the second cervical ganglion was completely necrotic. The geniculate ganglion, however, was normal, whereas the facial nerve was heavily infiltrated with lymphocytes.

From this case Denny-Brown et al. concluded "that damage to the seventh nerve can occur in the course of herpes zoster by the mechanism of an independent motor neuritis not directly related to inflammation of any ganglion." It should be remembered, however, that the skin of the external ear is supplied by the 5th, 7th, 9th, and 10th cranial nerves as well as the 2d and 3d cervical nerves with some overlapping. Anastomoses are present between the facial nerve and the other nerves mentioned. Denny-Brown et al. unfortunately failed to examine the 9th and 10th cranial sensory ganglia, which, as pointed out by O'Neill "they had reason to believe, from the occurrence of a circumscribed microglial reaction in the lateral portion of the medulla, were singly or doubly involved." Therefore their conclusion, cited above, is open to discussion.

Findlay's and the present case seem to be the only two of a clinically "pure" Hunt's syndrome with facial paralyses histologically examined.

Findlay concluded, that the pathologic findings in the geniculate ganglion in his case, in which most of the nerve cells were well preserved, were consistent with a history of herpes of the geniculate ganglion.

To us this conclusion seems unjustified.

As in our case only the temporal bone was available for histological examination, very few deductions can be attempted. One thing, however, is perfectly clear: The herpetic eruption was strictly localized to the "geniculate zone," a typical "Hunt's syndrome," and it was not due to a lesion of the geniculate ganglion.

Whether the herpes was due to a necrosis in some ganglion of another of the nerves which supplied the external ear with sensory innervation which we unfortunately had not the opportunity to examine, or whether it was caused by an independent motor neuritis of the facial nerve in which we found a heavy lymphocytic infiltration, it is impossible to say. According to Wohlwill such an independent motor neuritis may account for a herpes zoster without the finding of ganglion involvement.

One conclusion, however, is justified from our report: A typical "Hunt's syndrome" may be found without damage to the geniculate ganglion, and it supports O'Neill's statement: ". . . the pathologic grounds for incriminating the geniculate ganglion in dural herpes zoster are almost nonexistent."

For this reason, as stressed by Tschiassny, it does not appear justifiable to postulate histopathologic changes in the geniculate ganglion as a requirement for the diagnosis of the clinical entity "Ramsay Hunt's syndrome."

In one respect the four cases described above were in complete accordance: A heavy lymphocytic infiltration was demonstrated in each facial nerve. This is in striking contrast to the microscopic findings in Bell's ischemic palsy, where only vascular alterations and degenerations were found in the facial nerve, but never signs of inflammation (Kettel).

Conclusion

Denny-Brown, Adams, and Fitzgerald stated: Analysis reveals that the evidence for geniculate ganglionitis in the "Ramsay Hunt syndrome" is invalid.

We agree completely with these authors.

Summary

A woman, 78 years of age, who for six weeks prior to her death had suffered from a typical herpes zoster oticus (Ramsay Hunt's syndrome) with complete facial palsy and auditory symptoms, died from embolus of the pulmonary artery. Histologic examination showed that the geniculate ganglion was very little affected, whereas a heavy lymphocytic infiltration of the intratemporal part of the facial nerve was demonstrated.

These findings confirm the statement of Denny-Brown, Adams, and Fitzgerald, that the evidence for geniculate ganglionitis in the "Ramsay Hunt syndrome" is not satisfactory.

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Peripheral Facial Palsies Due to Tumors

Pathology and Clinical Picture: A Review of the Literature and a Report of Three Cases of Intratemporal Tumors of the Facial Nerve

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Tumors causing a facial palsy may originate either from the facial nerve itself or from the acoustic nerve, with which the facial nerve is intimately connected intracranially. They may also arise from the surrounding tissues along the entire length of the facial nerve, producing a facial palsy either by compression of the nerve or by infiltration, according to the site and nature of the tumor.

General Pathology

The pathology of tumors of peripheral nerves has been described in a paper by Busch and Christensen on which the following *general* account is mainly based, partly by direct quotation.

A peripheral nerve and its sheaths are formed in part from ectodermal tissue (axis cylinders, myelin sheaths, Schwann sheaths), and in part from mesodermal (epi-, peri-, and endoneurinum), and it is of fundamental importance to understand this structure in order to know which of its elements may form the point of origin for tumors.

According to their origin, Gagel divides the tumors of the peripheral nerves into two main groups:

I. Tumors Consisting Entirely of Ectodermal Tissue or of Both Ectodermal and Mesodermal Tissue.—1. Neurinoma or Schwannoma: This is the designation of a peculiar tumor arising from the cells of the sheath of Schwann, specifically the primitive stages of the latter, and therefore the tumors are ectodermal in origin. They are composed partly of Schwann cells and partly of connective tissue, whereas they do not contain true neural tissue. A more detailed histologic description is given in works by Verocay, Korbsch, Gnassi and Barrone, and more particularly, by Antoni, who, in a larger monograph, accounts for their origin and structure.

Antoni regards this tumor as preponderantly ectodermal, originating from the cells of Schwann, with a characteristic histological structure. Elongated fibril-forming cells poor in protoplasm lie in streaks crossing each other in various directions, and contain rod-shaped dark nuclei, which have a tendency to palisade arrangement. The vascularization is scanty and the connective tissue stroma is of variable abundance. These tumors also show a tendency to degeneration, through which the tissue becomes reticular.

2. Recklinghausen's Multiple Neurofibroma: A number of the peripheral tumors often contain an admixture of collagen connective tissue, which is often present in large quantities, for which reason the term fibroneurinoma, coined by Verocay, ought to be adopted as the proper designation of these tumors, instead of neurofibroma (von Recklinghausen), as these tumors do not contain true neural tissue.

Tumors made up entirely of ectodermal tissues are neuroepithelioma, sympathogonioma, sympathoblastoma, ganglioneuroma, and paraganglioma. In relation to facial palsy they are of no interest.

II. Tumors of Mesodermal Origin.—1. Perineural Fibromas, or Neurofibromas: benign tumors made up of connective tissue enveloping the nerve.

2. Plexiform Neurofibromas (Verneuil).

3. Sarcomas in various forms: fibrosarcoma, spindle-cell sarcoma, and polymorphocellular sarcomas with or without myxomatous degeneration.

Neurogenic sarcomas originating from the mesodermal sheath first appear as slowly growing, circumscribed, firm, movable tumors of varying histological structure. According to the histological picture, the sarcomas may be divided into three degrees of malignancy, even though there will also be transitional forms (Quick and Cutler). The malignancy increases with an increasing number of tumor cells and a decreasing proportion of connective tissue.

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The tumor may attack cranial nerves as well as peripheral ones, being sometimes solitary, sometimes multiple. Of the cranial nerves, only the olfactory and optic seem to escape.

The posterior roots are attached with a rather remarkable frequency, and tumors of the cranial nerves seem to show a preference for the unmixed nerves, especially the acoustic. Tumors may, however, grow also from mixed nerves, and will then commonly be found on the sensory root.

Classification of Material

According to the site of the tumor, the material may be divided clinically into intracranial, intratemporal, and extratemporal tumors, which will be dealt with separately.

Intracranial Tumors

According to Courville, neurofibromas of the acoustic nerve, epidermoid tumors, hemangioendotheliomas, meningiomas, osteosarcomas, and metastatic tumors are found along the petrous bone and may produce facial palsy. Roentgenograms reveal extensive irregular destruction of bone.

The principal tumors, the acoustic neurinomas and the epidermoid tumors, must be dealt with in some detail.

Acoustic Neurinomas.—These tumors are by far the commonest among those neoplasms which may cause a facial palsy.

A description of the pathology, symptomatology, diagnosis, and therapy of acoustic neurinomas is outside the scope of this paper, and references should be made to textbooks of neurology and neurosurgery. A very clear and comparatively short description has recently been published by Pool and Pava, from which most of the following details are cited.

In gross appearance acoustic neurinomas are discrete encapsulated fibrous tumors of the acoustic nerve that vary both in size and appearance, depending upon their growth characteristics and their age at the time of discovery. An acoustic neurinoma is usually round or oval, with a maximal diameter of 1 to 5 cm.

Bony erosion of the internal auditory meatus is often present because of tumor growth within the auditory canal in which these tumors may arise or extend for a variable distance.

The fifth as well as the seventh and eighth cranial nerves are generally compressed and distorted by large neurinomas, whereas the 9th, 10th, and 11th cranial nerves are not apt to be significantly affected.

Too frequently the facial nerve is attenuated into thin, flattened strands that cannot be separated from the tumor without injury or tearing. If the total removal is to be accomplished in such cases, it is usually necessary to sacrifice the facial nerve at this point. In some cases, on the other hand, the facial nerve can readily be peeled off the tumor, although even this may lead to temporary and occasionally to permanent facial paralysis.

In spite of the contiguity of the facial and auditory nerves, seventh nerve symptoms are neither common nor early.

According to Pool and Pava, who surveyed the literature, facial symptoms were present in 13.0% to 18.8%, whereas facial signs (facial weakness) were considerably more abundant, present in 48.3% to 65% of the series published.

For otologists it is of the utmost importance to remember that a unilateral deafness must arouse the gravest suspicion of an acoustic neurinoma. Among Pool and Pava's 122 cases, unilateral deafness was noted by 92.0% of the patients and total or partial deafness was by examination demonstrated in 98.3%.

Impaired unilateral function of the labyrinth was found in 98.9% of the cases. Olivecrona demonstrated deafness in 99.2% and impaired labyrinthine function in 90.8% of his cases.

As stated above, acoustic neurinomas may cause a facial palsy, but most of the palsies connected with these tumors arise

postoperatively, and consequently belong to the group of traumatic palsies.

Epidermoid Tumors.—Epidermoids (cholesteatomas) of the petrous bone are best dealt with at this point because they may break through bone and invade the cranial fossae. Pennybacker has described three cases in which facial palsy came on gradually, accompanied by deafness of the homolateral ear of a predominantly middle-ear type, and loss of caloric responses. Otherwise the neurological and ophthalmologic examination as well as the spinal fluid were normal.

The operative finding was similar in all three cases: "There was erosion of the upper surface of the petrous bone by a soft, flaky white mass, creating a cavity in which the facial canal was bared between the internal acoustic meatus and the genu, and the nerve was flattened."

Jefferson and Smalley reported six cases of epidermoids of the temporal bone, which will be described later. In one of them the tumor was localized to the petrous part of the temporal bone, and was identical with those described by Pennybacker.

Miller and Uihlein described a case in which a man 30 years of age had suffered for some years from intermittent facial palsy and impaired hearing of the conduction type. Roentgenograms showed decalcification and erosion of the tip of the right petrous bone and questionable enlargement of the right internal auditory meatus.

Through a temporal craniotomy a round mass approximately 1 cm. in diameter with the characteristic pearly appearance of its capsule was seen medial and slightly posterior to the Gasserian ganglion. This tumor appeared to lie along the petrous ridge, and extended into the posterior fossa.

The aural symptoms are thus very similar to those caused by an acoustic neurinoma, but, whereas facial palsy is a rather rare symptom preoperatively in acoustic neurinomas, it was a constant symptom in epidermoids of the tip of the petrous bone, as described by Pennybacker.

Even though in acoustic neurinomas the albumin content of the spinal fluid and the intracranial pressure are usually increased, and abnormalities may be discovered by ophthalmologic examination, the lack of these symptoms does not exclude the possibility of an acoustic neurinoma. In cases of doubt, ventriculography may be indicated. Straight roentgenograms, however, may be very helpful. In Pennybacker's case an erosion of the upper surface of the petrous bone was seen, quite different from the enlargement of the internal acoustic meatus generally encountered in acoustic neurinomas.

All the patients described above recovered except for facial palsy, which persisted in two of Pennybacker's three patients.

Sheridan and Banham described a case of an uninfected epidermoid, arising from a congenital epithelial implant.

The patient, 35 years of age, had been deaf in the right ear since childhood and had for the last four months suffered from a complete homolateral facial palsy.

The epidermoid had completely replaced the inner ear and extended medially as far as the internal auditory meatus. The facial nerve was seen running through the tumor in its intratympanic course.

If the facial nerve must be sacrificed in removing the acoustic neurinoma, the nerve should be grafted according to the method of Dott. If the facial nerve is saved but reinnervation of the muscles does not occur, Dott's operation should be performed at a later date. If this fails, an anastomosis operation should be done, and if this intervention also proves unsuccessful, plastic operations should be resorted to.

Intratemporal Tumors

Within the temporal bone a facial paralysis may arise either from tumors originating from the facial nerve, which will be described in detail, or from the surrounding tissues, which will be dealt with only under the heading Differential Diagnosis.

Tumors of the Facial Nerve.—Within the temporal bone neurinomas, fibroneurino-

mas, and sarcomas originating from the facial nerve have been described.

For practical reasons the "pure" neurinomas as well as the fibroneurinomas will be dealt with under one heading: Neurinomas.

Neurinomas: To this group belong the majority of tumors, 32 cases having been reported, including the 3 described below.

As the development of a neurinoma on a cranial nerve containing only motor fibers is of extremely rare occurrence, the relative frequency with which they are found on the facial nerve is a striking feature.

However, the facial nerve is a mixed nerve, and neurinomas on it should probably be regarded as examples of neurinomas originating from the sensory fibrils of mixed cranial nerves (Kettel, Loeliger).

Solitary neurinomas are histologically of a benign nature. They grow slowly by expansion in the direction of least resistance.

In the petrous and vertical segments of the Fallopian canal the bony walls are more resistant to expansion than in the horizontal segment. Hence the onset of paralysis may be earlier with a tumor in either of the former locations than with one in the latter location. (Bogdasarian)

As they increase in size, they tend, as stressed by Roberts "to invade adjacent tissues, and may even destroy vital structures and in this sense may become malignant." Often they are firm and well encapsulated, and consequently easy to remove, but in some cases the tumor is soft or cystic and degenerative changes may occur.

The symptoms caused are directly dependent upon the site of the tumor and the direction of its growth.

In reviewing the literature (July, 1958), 29 reports of intratemporal neurinomas or fibroneurinomas of the facial nerve have been found, to which by courtesy of Cawthorne and Lundgren 3 cases are added below, 32 cases in all.

Grafton Love has described a case of a fibroneurinoma within the petrous bone near the internal acoustic meatus, accompanied by unilateral deafness and facial palsy. Although according to the author the tumor presumably took its origin from the acoustic nerve, this could not be proved, and therefore it is not included in the present report.

The same applies to a traumatic neurofibroma of the facial nerve described by Eigler. It resulted from a fracture of the temporal bone with a lesion of the facial nerve. According to the author it was no "true" neoplasm, but corresponded to the neuromas seen on regenerating nerves previously severed.

According to the point of origin, the tumors may be divided into two groups, arising from (*a*) the vertical, and (*b*) the horizontal segment of the facial nerve respectively.

I. Neurinomas arising from the vertical segment of the facial nerve. This is the most frequent site. These tumors are located on the nerve in its course between the bend at the posterior end of the lateral semicircular canal and the stylomastoid foramen.

To this group belong 23 cases, including the 3 reported below. The first was published in 1931 by Schmidt; the others, by Schroeder, Grossmann and Leidler, Altmann (5 cases), Lemaitre and Broutmann, Ohnishi, Williams and Pastore, Kos, Bloch and Aboulker, Wysotskaja, Skoog, Cawthorne (2 cases), Kettel, Lundgren (3 cases), Collins and Thomson, Rosenwasser.

Cawthorne and Lundgren gave me permission to report and include the following cases in this survey:

CAWTHORNE'S CASE 1.—Woman 19 years of age. No history of previous attacks of facial palsy or of any serious illness apart from childhood ailments.

A facial palsy on the left side had started gradually seven months before admission and been complete for the last four months. There was loss of taste on the left side of the tongue anteriorly. Lacrimation was normal on both sides. The central nervous system was normal. No deafness, no vertigo.

The facial palsy was complete and so was the reaction of degeneration. The drums and hearing were normal on both sides. Mastoid x-rays showed

cellular mastoids, without blurring of the cells, and with no evidence of bone destruction.

At operation an almost round swelling 1 cm. in diameter was found in the continuity of the facial nerve starting 5 mm. above the stylomastoid foramen. The swelling was removed, leaving a gap in the facial nerve of 9 mm. A nerve graft was inserted and facial nerve function restored.

Histological report: fibroneurinoma.

CAWTHORNE'S CASE 2.—Boy aged 8 years.

For 18 months there had been increasing weakness of the right side of the face, and for 6 months there had been a complete palsy.

During the last two months deafness in the right ear had been noted, and an otologist had found a red swelling in the posterior part of the right tympanic membrane. This had been incised, and after this there was slight but continuous ear discharge.

On admission there was complete facial paralysis on the right side with loss of taste, but no loss of lacrimation. No hearing on right side. Other cranial nerves were normal. X-ray showed some bone destruction in the mastoid on the right side only.

At operation the mastoid had been partly excavated, and the mastoid, aditus ad antrum and middle ear were full of what appeared to be pale granulation tissue. On clearing this away it was found to be spreading from the trunk of the facial nerve in the whole of the descending portion. The gap in the continuity of the nerve trunk was too large for a nerve graft, and so a faciohypoglossal anastomosis was performed.

Histological report: fibroneurinoma.

LUNDGREN'S CASE 3 (The first two have been published previously).—Woman 39 years of age. On admission the palsy was not complete and the sense of taste was normal. A whitish tumor was seen in the external auditory meatus, but the upper part of the drum was visible, red, and infiltrated. The hearing was reduced to 1 meter for spoken voice.

Roentgenograms showed a vast localized destruction just below the posterior semicircular canal, reaching from the facial canal to the sigmoid sinus.

At operation a tumor the size of a hen's egg was found originating from the denuded vertical segment of the facial nerve, having extensively exposed the dura in the posterior cranial fossa, penetrating below the posterior semicircular canal and reaching the bulb of the jugular vein. The facial nerve had been almost completely destroyed; only a few strands remained and later they necrotized.

Histological examination showed a neurinoma.

As already described, the neurinomas grow slowly and by expansion, in the direction of the least resistance. The objective findings depend largely on the stage of the disease at which the patients first appear for examination.

To give an exact pathologic and clinical picture of the neurinomas of the facial nerve based upon the material available is, I regret to say, impossible, as many of the reports are rather incomplete. This implies that in a few cases it has been hard to determine whether the tumor had arisen from the vertical or the horizontal segment of the nerve.

These exceptions, however, are so few, that they do not interfere with the general evaluation of the material.

Surveying the available reports, the pathologic findings at the time of operation may be divided into approximately three groups, which, however, only represent three different steps in the development and growth of the tumor.

1. Tumors restricted to the mastoid process itself, without appearing in the external auditory meatus. To this group only two cases belong, Cawthorne's Case 1 (described above) and that of Kettel.

In the latter, as described in a previous paper, a destruction of the mastoid process was found, extending from the posterior end of the lateral semicircular canal to the stylomastoid foramen, while posteriorly it fell just short of the sinus. It was filled with grayish-red masses. (Microscopy: neurinoma.) The tumor originated from the completely denuded vertical segment of the nerve, the posterior wall of the facial canal having been destroyed.

In both these cases, the last one of seven years' duration, the external acoustic meatus, and especially the membranous lining of the posterior wall, the drum, and the hearing were normal.

2. Tumors restricted to the mastoid process and at the same time appearing in the external acoustic meatus. This finding is much commoner. The first sign of an imminent destruction of the posterior osseous wall is a reddish swelling of the latter, feeling soft to touch of a probe (Schmidt, Ohnishi).

Later in development the tumor will appear as polyps, bearing a disconcerting resemblance to the polyps seen in a chronic otitis. When removed, the drum in most cases looks normal. The polyps may grow so large that they obturate the external acoustic meatus completely, or they may grow so exuberantly, in clusters like a bunch of grapes, that they occupy all available space in the cavum conchae (Williams and Pastore).

3. Tumors invading the middle and internal ear and the cranial fossae by expansion. Neurinomas arising from the vertical segment of the facial nerve may at the same time destroy the mastoid process, and the posterior osseous wall, appearing in the external acoustic meatus and invading the middle ear (Cawthorne, Schroeder, Williams and Pastore), the labyrinth (Grossmann and Leidler, Schroeder, and Rosenwasser), and the cranial fossae, giving rise to an abscess of the brain (Schroeder) or even destroying the anterior wall of the external acoustic meatus and invading the parotid gland (Grossmann and Leidler).

By secondary infection the tumors give rise to a foul-smelling discharge and to abscesses in the organs invaded.

Thus it will be seen that tumors arising from the vertical segment of the facial nerve, which in the beginning are restricted to the mastoid process or perhaps appear in the external auditory meatus as polyps, may in time by expansion invade the middle and internal ear, may expose the dura in the posterior and middle cranial fossae, and even invade the brain.

It should also be stressed that the drum is quite normal and not affected, even if in some cases it is concealed by a polyp, until the middle ear is finally invaded by the tumor.

II. Neurinomas arising from the horizontal segment of the facial nerve. To this group belong nine cases (Greifenstein, Fehre, Roberts, Bogdasarian, Loeliger [2 cases], Rejtö, Rainer, Graf).

Tumors may arise either from a nerve normally enclosed in the osseous facial canal and then break through the thin brittle bone of the Fallopian canal, or they may arise at a place where a dehiscence in the canal is present. From this site of origin the tumors may grow in all directions. The middle ear is inevitably invaded, in contrast to cases of neurinomas originating from the vertical segment of the nerve, where, as just described, the drum is not affected until a late stage of the disease, if at all.

In the majority of these cases the drum is found to be red and bulging, very much as in an otitis media. Because of the early development of the tumor within the tympanic cavity, and subsequent impairment of hearing, the majority of these patients apply for treatment at a time when the process is localized to the middle ear and before it has broken through the drum. In Bogdasarian's case tumor masses prolapsed into the external auditory meatus on incision of the bulging drum. Only three patients (Roberts, Rejtö, Loeliger's Case 1) were first seen after the tumor had invaded the external auditory canal and they were not examined till 30, 12, and 4 years respectively after onset of the disease.

Loeliger had the opportunity to follow the whole sequence of events. He examined his patient one year after the onset of an increasing deafness, and noted a peculiar reddishness and bulging of the drum.

On paracentesis there was profuse hemorrhage. Not until three years after incision did facial weakness begin. The patient reappeared seven years after the onset of hearing impairment, and at this date an obturating tumor was seen in the external auditory meatus.

The tumor may, however, not only grow outward through the drum, but also in other directions, and the pathologic picture which presented itself when the patients first applied for examination and treatment may be surveyed as follows:

1. Tumor restricted to the tympanic cavity. In one case (Fehre) a small neurinoma of the facial nerve was observed casually at dissection, whereas the tympanic cavity was free. In another case a solitary tumor was visualized through a transparent drum (Bradley and Maxwell). However, in the majority of cases clinically observed the tumor had arisen from the horizontal segment and filled the tympanic cavity (Greifenstein, Bogdasarian, Rainer, Graf).

2. Tumors invading the tympanic cavity, the mastoid process, and the cranial fossae. Just as tumors originating from the vertical segment of the nerve may by expansion invade the middle ear, so tumors originating from the horizontal segment may invade and fill the mastoid process (Roberts, Loeliger's Case 2, Rejtö).

Tremble and Penfield have reported a case in which by a beautiful topognosis a diagnosis of a tumor at the site of the geniculate ganglion was arrived at.

> At a point about 4 mm. from the bend and just above the oval window, the nerve was involved by a vascular mass of tissue, which proved to be a new growth. On removal of the roof of the middle ear, a tumor herniated into the opening. It had extended through the bone from the facial canal to the subdural space apparently following the canal of the greater superficial petrosal nerve. The main portion of the tumor thus presented beneath the dura on the anterior aspect of the petrous pyramid.

Thus it will be seen that tumors arising from the horizontal segment of the nerve are primarily restricted to the middle ear, and early give rise to bulging of the drum, which may become an angry red. Not until a later date are the external auditory meatus and the mastoid process invaded by expansion of the tumor.

Sarcomas: Sarcomas arising from the facial nerve are extremely rare, only two cases having been described.

Kettel (1950) reported a case in a woman 57 years of age, who for 14 months had suffered from an increasing facial palsy, which was complete on admission. The external acoustic meatus, the drum, and the hearing were normal, and the rest of a very careful examination, including x-rays of the temporal bone, showed nothing abnormal.

At operation a reddish irregular solid tumor, the size of a small hazelnut was discovered, having destroyed the posterior osseous wall of the Fallopian canal, from a few millimeters below the posterior end of the lateral semicircular canal to a few millimeters above the stylomastoid foramen, and expanding posteriorly into the mastoid cells. The tumor originated from the vertical segment of the facial nerve, having completely infiltrated the stem of the nerve. It was resected, and a nerve graft inserted. Microscopy showed a neurogenous spindle-cell sarcoma.

The second case was published by Guttman and Simon (1951). A man 43 years of age had for nine years complained of increasing facial palsy, which was now complete, as well as discharge, swelling, and deafness in the homolateral ear, coming on painlessly. A radical mastoidectomy had been done four years previously without any effect on the paralysis, discharge, or deafness. One year before admission the patient had noted a progressive soft, nontender swelling about the ear.

The external auditory meatus was filled with seropurulent discharge and grayish-pink granulation tissue, the hearing greatly reduced.

The mastoid cavity was filled with pearly grayish-blue highly vascular, exceedingly friable tissue. This was firmly attached to the dura in the posterior and middle cranial fossae, continuing down beneath the mastoid, and extending anteriorly to just in front the ear.

Microscopy showed neurofibrosarcoma.

Four years later the patient returned, complaining of headache and impairment of hearing. There was an increase in swelling about the mastoid and bilateral papilledema. A neurosurgeon found an enormous growth involving most of right cerebellar dura, which he removed. Microscopy showed a fibrosarcoma of low-grade malignancy.

Four years later his only complaint was the facial palsy. The external auditory canal had healed.

This case goes to show that, even if the neurosarcomas are histologically malignant, the degree of malignancy is so low that clinically they behave just like the benign neurinomas.

Symptomatology

It has been maintained that the picture of neurinomas arising from the vertical segment of the facial nerve is a clinical entity (Altmann, supported by Guttman and Simon, Roberts).

This, however, does not hold true any longer. Altmann (1935) based his description upon four cases of his own and two from the literature, and in all cases the tumor had arisen from the vertical segment.

Later, in studying the 32 reports now available, we have learned that they may also originate from the horizontal segment and I quite agree with Graf that the clinvariations are primarily explained by the ical picture is not a single entity, and that site of origin of the facial neurinoma, and the direction of its later development.

Consequently, in trying to outline the clinical picture as we know it today, it is imperative to distinguish between neurinomas arising from the vertical and from the horizontal segment respectively.

We have information about the age in 29 cases: 3 patients were younger than 20 years, 19 between 30 and 39, and 7 were 40 years old or more. Sex is stated in 27 cases, and 20 of these were women.

Thus it will be seen, that neurinomas of the facial nerve are found especially in women between the age of 20 and 39. In this respect there is no difference between neurinomas arising from the vertical and from the horizontal segment of the nerve.

Clinical Picture of Neurinomas Arising from the Vertical Segment of the Facial Nerve

The group of neurinomas arising from the vertical segment of the facial nerve comprises 23 patients.

Unfortunately it is impossible to give an exact description of development and combination of symptoms because some of the reports are rather incomplete. The following symptoms present on admission will be surveyed separately.

1. *Facial Palsy.*—In nearly all the cases this was the first symptom to appear, and with a few exceptions the palsy was complete on admission, having lasted from 1 to 15 years, in one case even longer.

Generally the palsy increases gradually over months, but it may come on quite suddenly (Schmidt, Kettel). Remissions are seldom noted but they may take place (Kettel).

Even if facial palsy is generally the first, and often for years the only symptom, it may be preceded by aural symptoms, and, what is most important, a palsy may be completely lacking. In Lundgren's Case 1, described previously, the patient had complained only of impaired hearing and tinnitus. A neurinoma the size of a hazelnut was removed from the anterior side of the vertical segment of the nerve, but neither before nor after operation was there any trace of facial weakness. According to Lundgren there was no palsy in Skoog's case either.

2. *Aural Symptoms.*—In some cases of neurinomas arising from the vertical segment of the facial nerve, aural symptoms may be present, generally at a late date. As described, facial palsy in the majority of cases started before aural symptoms were noted.

Impaired hearing may, however, be the first symptom of a neurogenous tumor, as, e. g., in Lundgren's Case 3, in which facial weakness started six months later, or it may be the only subjective symptom, as in Lundgren's Case 1, where any signs of facial weakness were lacking just as in Skoog's case.

As long as the external auditory meatus is clear (Cawthorne, Kettel), or a polyp does not completely obstruct the external canal (Lundgren, Collins and Thomson),

the hearing is normal. If it is completely blocked, the hearing will be impaired (Altmann, Grossmann and Leidler) for airborne sounds, and, if the tumor invades the middle or internal ear, deafness may result (Schroeder, Lemaitre and Broutmann, Williams and Pastore, Cawthorne's Case 2, Altmann).

Discharge from the external canal may result from secondary infection of the middle ear after the tumor has invaded the tympanic cavity.

Vertigo may result from arrosion of the lateral semicircular canal (Altmann, Rosenwasser).

3. *Pain.*—Altmann states that severe pain is totally absent, and some authors claim that these cases are absolutely painless.

Even if this applies to the majority of the cases, exceptions to this rule have been reported (Grossmann and Leidler, Williams and Pastore, Kettel).

In Kettel's case, in which the tumor was restricted to the mastoid process, the patient had complained for seven years of pains in the mastoid region. The pain disappeared in immediate conjunction with removal of the neurinoma.

In Williams and Pastore's case pains were also present, but here the tympanic cavity and the pyramid had been invaded and infected.

4. *Cerebral Symptoms.*—These have been observed in only one case (Schroeder) in which the brain was invaded by the tumor. It should be remembered, however, that exposure of the dura in the posterior cranial fossae by tumor masses is not an uncommon finding in more advanced cases.

5. *Polyps in the External Auditory Meatus.*—This is a common symptom. However, in a few cases (Cawthorne, Kettel) the external auditory meatus as well as the drum were completely normal.

In Schmidt's and Ohnishi's cases a reddish swelling only was observed at the posterior osseous wall, 0.5 cm. lateral to the drum, feeling soft to the touch of a probe. There was no polyp.

The polyps may appear as round smooth tumors or they may be more irregular in appearance.

Thus Altmann's view, that polyps always are present in the external auditory meatus, cannot be sustained. Nor can his assumption that it is doubtful whether the facial palsy occurs before the tumor can be seen in the meatus, as in the cases of Cawthorne and Kettel the palsy was complete but there were no polyps in the external canal.

Clinical Picture of Neurinomas Arising from the Horizontal Segment of the Facial Nerve

The group of neurinomas arising from the horizontal segment of the facial nerve comprises nine patients.

In this group the middle ear is inevitably invaded from the start, in contrast to cases of neurinomas arising from the vertical segment, and this explains the difference in the clinical picture in the two groups. In two cases (Greifenstein, Fehre) the neurinoma was found by dissection and the clinical symptoms not recorded in detail. To this group belong another seven cases (Roberts, Bogdasarian, Loeliger, 2 cases, Rainer, Rejtö, Graf).

1. *Facial Palsy.*—In six cases the palsy was complete on admission, in one partial (Loeliger's Case 1), whereas in one case in which the neurinoma completely filled the middle ear facial palsy had not been noted (Greifenstein).

The palsy generally arose gradually and remained permanent, but in one case (Graf) the palsy improved during six years of observation and then suddenly became complete again.

In two cases (Loeliger's Case 2, Rainer) facial palsy was preceded by facial tic.

2. *Aural Symptoms.*—In neurinomas arising from the vertical segment, facial palsy was in nearly all cases the first and often for years the only symptom, whereas impairment of hearing was a late symptom, and in many cases was completely lacking.

In tumors arising from the horizontal segment of the facial nerve, on the other hand, impaired hearing of the conductive type is an early symptom, which in most cases preceded facial palsy, often by several years.

As pointed out by Bogdasarian, "deafness of varying degrees and types occurs as the first presenting symptom, depending on whether the disturbance affects the movements of the ossicles or leads to fixation of the footplate of the stapes or occlusion of the round window."

In all cases in which hearing was recorded an impairment was noted, and two patients were deaf (Roberts, Loeliger's Case 2).

Discharge by secondary infection of the middle ear is not uncommon, and vertigo was observed in one case, in which a fistula on the lateral semicircular canal was found at operation.

3. and 4. *Pains and Cerebral Symptoms.* These have not been described in this group.

5. *Polyps in the External Auditory Meatus.*—These were seen in only two cases (Loeliger's Case 1, Rejtö) and at a late stage. On the other hand, bulging and swelling of the drum is an early symptom, as has been pointed out.

Comments

As has been explained, in trying to establish the clinical picture of neurinomas arising from the facial nerve, it is imperative to distinguish between those originating from the vertical and those from the horizontal segment. This distinction applies especially to the early stages of the neoplasms which it is so important to recognize in order to achieve good therapeutic results.

In the end, however, the clinical picture will be the same for both types, when tumor masses, regardless of their site of origin, have destroyed the middle ear and the mastoid process.

The early picture may be summed up as follows:

1. The tumor appears chiefly in women between 20 and 39 years of age.

2. Facial nerve.

(*a*) In tumors arising from the vertical segment, facial palsy is generally the first and often for years the only symptom. In two cases, however, there was not a single trace of facial palsy. (*b*) In tumors originating from the horizontal segment, facial palsy may still be the first symptom, but in the majority of cases it is preceded by hearing impairment. Facial tic may start before facial weakness can be observed. In both groups the palsy generally comes on gradually and remains permanent, but the onset may be quite sudden, and remissions have been observed.

3. Aural symptoms.

(*a*) In tumors arising from the vertical segment hearing may be completely normal. It may sometimes be necessary to remove an obstructing polyp from the external acoustic meatus. (*b*) In tumors originating from the horizontal segment hearing impairment is an early symptom, and generally it precedes facial palsy, often by years.

Hearing impairment in both groups is initially of the conductive type, but deafness may also result from destruction of the labyrinth.

Discharge may follow a secondary infection of the middle ear, when the tympanic cavity has been invaded by tumor masses.

Vertigo in both groups is a rare symptom and always late in onset. Fistulas have been observed in both groups at operation at the lateral semicircular canal.

4. Pain. This is in both groups a rare symptom, but it has been observed.

5. Cerebral symptoms. These are seldom observed and, if present, are always seen at a late date after invasion of the brain. The dura in the middle and posterior cranial fossae have in both groups often been exposed by the tumor.

6. Polyps in the external auditory meatus.

(*a*) In tumors arising from the vertical segment, polyps in the external auditory meatus were seen in the majority of cases when the patients first applied for treatment. The canal may, however, be perfectly clear. When removed, the drum looks normal if the middle ear has not been invaded, which generally happens only at a late date. (*b*) In tumors arising from the horizontal segment, polyps have been observed in only two cases, and those at a late date. The drum, on the other hand, is in the early stages red, swollen, and bulging, and on myringotomy a resistance is felt and lively bleeding follows. A prolapse of tumor masses into the canal may follow incision.

Diagnosis

Tumors Arising from the Vertical Segment of the Facial Nerve.—The most im-

portant factor in establishing the diagnosis is simply to remember that a tumor of the nerve may be the cause of this palsy.

A preoperative diagnosis has been made in a few cases (Bogdasarian, Bradley and Maxwell, Lundgren) but in the majority of cases the indication for operation has been suspected Bell's ischemic palsy or chronic otitis media.

If the palsy comes on gradually and painlessly, as the only symptom, and without subsequent improvement, a tumor of the vertical segment of the nerve should be seriously suspected. A roentgenogram of the mastoid process should always be taken. A blurring of the mastoid cells in a patient with facial palsy, without a history of middle-ear infection, and with a completely normal drum and hearing, is suspicious of a tumor of the vertical segment of the nerve. Even more suspicious is a well-defined destruction of the mastoid cells near the Fallopian canal (Graf, Cawthorne). Tumors of the nerve may also cause erosion of the posterior inferior wall of the external acoustic meatus, of the floor of the tympanic cavity, or of the lateral attic wall (Altmann).

A dilatation of the stylomastoid foramen has been seen in cases in which the tumor affected the facial nerve outside as well as inside the Fallopian canal (Maxwell, Graf).

It should be strongly emphasized, however, that the onset of the palsy may be quite sudden, that remissions may take place, that pains may be present, and that roentgenograms may be completely normal.

In such cases it is quite impossible to make the differential diagnosis between a case of Bell's ischemic palsy and a neurogenous tumor, and an explorative operation is indicated.

In most cases a polyp was seen in the external auditory meatus when the patient first applied for treatment. A biopsy, which in some cases must be repeated, will establish the diagnosis.

Tumors Arising from the Horizontal Segment of the Facial Nerve.—Owing to the site of origin, the sequence of symptoms in tumors arising from the horizontal segment is generally quite different from that presented by tumors originating from the vertical segment.

The middle ear is involved at an early date and consequently the hearing is generally impaired before facial palsy begins, and often several years earlier.

The majority of patients presented themselves with hearing impairment, sometimes facial palsy, and a red, bulging, swollen drum.

Differential Diagnosis

Attention should be paid to epidermoids (cholesteatomas) of the petrous part of the temporal bone, to glomus jugulare tumors, to chronic otitis media, to tuberculosis, and to carcinoma of the middle ear.

As already described, Pennybacker has reported three cases of epidermoids, and Jefferson and Smalley have described six cases, of which one was localized as were Pennybacker's. Four of the remaining five involved the antrum (one the attic too) and petrous bone, the last the mastoid and antrum. On roentgenograms they formed "clearly defined cystlike spaces with an even capsule of condensed bone, very thin superiorly and apt to scale away on dissection, leaving the dura exposed."

The palsy in all cases developed gradually. In one of them a temporary improvement was noted, followed by relapse.

The hearing was impaired in all cases, and one patient was deaf.

Only slight alterations were noted at the drums and none of them were perforated.

On operation, the facial canal was in most cases found eroded, and the nerve compressed or severed.

The authors believe that most, if not all, of their series were of embryonal origin, and not secondary to epithelial invasion from without.

Glomus jugulare tumors are neoplasms of the carotid body type, arising from the glomus at the bulb of the jugular vein.

Lundgren has reported four cases of his own and discussed three cases, previously reported by Rosenwasser, Le Compte, and Kipkie. From the bulb of the jugular vein the tumors grow into the middle ear, and through the tympanic membrane into the external auditory canal, resembling ordinary polyps. They may, however, also grow into the mastoid process and the petrous bone.

The glomus jugulare tumors, or, as Lundgren prefers to call them, the tympanic body tumors, should be remembered because they may very rarely cause a facial palsy by invasion of the middle ear (Rosenwasser, Horn and Stout, Capps, Seiffert). Also, having perforated the drum, they may by appearance in the external acoustic meatus resemble neurogenous tumors. It should be stressed that extensive bleeding occurs if these tumors are traumatized.

Guild has shown that glomus formations may be present normally in locations other than that originally described, among others along Arnold's nerve as far peripherally as the vertical part of the facial canal.

This explains why Bradley and Maxwell in one case were able to find a glomus jugulare tumor located beneath the mastoid tip, involving the facial nerve at the stylomastoid foramen. Due attention should be paid to these tumors, which seem to appear comparatively often. Thus Capps has been able to collect quite a considerable series of his own (23 cases) within comparatively few years.

For detailed information regarding this category of tumors reference should be made to papers by Lundgren and Lattes and Waltner.

In advanced cases, in which the middle ear has been invaded by tumor masses and secondarily infected, the possibility of chronic otitis media, tuberculosis of the middle ear, and a carcinoma should be considered.

Roentgenograms may, as already described, be helpful. Opacity of the mastoid air cells on x-ray is indicative of a chronic otitis media, without excluding a neurogenous tumor.

In distinguishing between chronic otitis media and a neurogenous tumor, as well as a glomus jugulare tumor, Bradley and Maxwell palpated the membrane with a flexible silver probe. In a chronic otitis the drum felt fluctuating; in a neurogenous tumor and in a tumor of the glomus jugulare probing will reveal a rather rubbery firmness.

A diagnosis of tuberculosis is rendered less likely by the absence of the signs of tuberculosis elsewhere in the body.

In cases of carcinoma discharge from the ear is observed for a long time before facial palsy begins. In a neurogenous tumor, on the other hand, the palsy, if it occurs, is the first symptom in the vast majority of cases.

Pain is a frequent symptom in carcinoma of the middle ear, and is seldom seen in a neurogenous tumor, but it has been described.

In every instance one or repeated biopsies should be done; in the majority of cases they will reveal the cause of the palsy.

Prognosis

It is generally agreed that the prognosis quoad vitam is favorable. Some reservation would, however, be appropriate, as a tumor near the middle ear and the labyrinth implies the risk of complications from the latter, when the tumor grows into them.

This reservation is perhaps more theoretical than practical, and it must be admitted that some of these patients did suffer from labyrinthine as well as cerebral complications, and that none of them died from the disease. The most extensive destructions were seen in Schroeder's case (labyrinthine destruction, abscess of the brain), and nevertheless the patient was alive 16 years after operation, in spite of the im-

possibility of removing the tumor completely.

Prognosis for future function of the facial nerve, on the other hand, is extremely bad, if the nerve is not repaired at the site of lesion.

However, thanks to Bunnell and Ballance and Duel, it should today be easy to restore facial nerve function in the vast majority of these patients, as will be described below.

Therapy

Treatment is exclusively surgical.

The Tumor.—The tumor should be removed as soon as the diagnosis is established. I quite agree with Roberts that explorative operation is indicated when results of repeated biopsies and roentgen examinations are inconclusive.

In tumors arising from the vertical segment of the facial nerve, and restricted to the mastoid process, a simple mastoidectomy only is needed to remove the tumor. If the tumor has destroyed part of the posterior osseous wall, an atticoantrotomy will in many cases be sufficient for the removal of the tumor.

If, however, the tumor has advanced into the middle or internal ear, a radical mastoidectomy or a labyrinthectomy must be done.

Facial Palsy.—Removal of the tumor is absolutely indicated, but it is just as important to repair the facial nerve at the same session, by nerve grafting according to the method of Bunnell and Ballance and Duel.

Considering the fact that Bunnell published his papers in 1927 and 1931, that the fundamental work of Ballance and Duel appeared in 1932, and that all cases of neurinomas of the facial nerve (with two exceptions) have since been published, it is surprising to see that attempts at direct repair of the nerve have been made in only a few cases, by Cawthorne, Collins and Thomson, Cranmer, and Kettel.

Two conditions must be fulfilled to obtain a clinically satisfactory result by direct nerve repair.

1. The site of lesion must be accessible. This condition is fulfilled in facial palsies caused by intratemporal neurinomas, all of which have arisen between the geniculate ganglion and the stylomastoid foramen.

2. The muscles must not have degenerated at the time of repair. This depends upon how soon after onset of the palsy the patients arrive, or, in cases of long standing, upon the treatment which has been given to keep the muscles alive during the time of observation.

Repair of the nerve is absolutely indicated in all cases in which the muscles respond strongly to the galvanic current. If degeneration of the muscles has taken place, plastic operations should be resorted to.

In a previous paper I have reported the results of 78 attempts at nerve grafting and nerve suture. At that time repair had in six cases been performed only recently. Now treatment is finished in all the cases, and information is lacking about one patient only, a foreigner.

As described the material could be divided into: (*a*) unfavorable cases (14 patients, in which the operation either could not be completed for technical reasons to me unsurmountable, or partial degeneration of the muscles had taken place at the time of repair; and (*b*) favorable cases, in which the conditions for repair were good and the operation properly completed.

To the second group 64 patients belonged; 63 of these patients have been reexamined. In 57 cases, or 90%, a clinically satisfactory result was obtained: all patients looked normal with the face at rest; 6 patients could only smile; 44 patients could smile and screw the eye up; 7 patients could smile, screw the eye up and wrinkle the forehead a little.

In six cases complete or partial failure followed the attempt to repair the nerve. Among these were my two cases of tumors of the facial nerve, a neurinoma and a sarcoma, both of them arising from the vertical segment.

In the first case (the neurinoma) complete failure followed nerve grafting, due entirely to my own fault. The tumor had severed all the superficial fibers of the nerve; only some deep strands remained, which macroscopically looked normal.

I simply tried to appose the severed fibers, but reinnervation did not occur. When a year later a new attempt at grafting was made, there was no connection at all between the stumps of the nerve proximal and distal to the site of the tumor. A graft was now inserted, but at this late date the distal end of the nerve had probably been transformed into more or less dense fibrous tissue, unable to conduct downgrowing fibrils to the muscles.

In this case and, as reported elsewhere, in some other cases, where the nerve for other reasons had been partly damaged in a localized spot, so that only an atrophic strand connected the normal parts of the nerve proximal end distal to the site of lesion, I have made the unforgivable mistake of leaving this strand in situ, and just inserting a lateral end-to-side graft.

In such cases one should never be afraid to resect the damaged part of the nerve and insert a graft, because what may look like remaining neurofibrils able to resume function is in most cases only connective tissue.

In my second case, a sarcoma, the result of nerve grafting was that the mouth was straight and could be moved freely. However, the patient was unable to close her eye, and a partial tarsorrhaphy was done. The palsy was consequently noticeable with the face at rest, and the result was accordingly classified as clinically unsatisfactory. However, she is still alive nine years after the operation, without any sign of recurrence, and extremely satisfied with the result obtained, which is illustrated elsewhere. She had had an intensive course of x-ray treatment postoperatively, and perhaps this damaged the graft, and made it less suited as a path for the downgrowing neurofibrils.

If only nerve grafting is performed in time, and properly, the patients have at least a 90% chance of recovering clinically satisfactory function of the facial nerve, and with a better technique (all my failures were among my early cases) the results may be even better.

It is my opinion that in years to come the prognosis for the future function of the nerve is going to be just as good as the prognosis for life in cases of intratemporal tumors of the facial nerve.

Extratemporal Tumors

Tumors of the Facial Nerve.—Three cases of cirsoid neurofibroma of the facial nerve have been recorded by Maxwell.

The first patient, a man 27 years of age, had for four years noticed a small hard oval mass at the angle of the jaw, but there had been no pain or facial palsy, and no symptoms referable to the ear.

At operation the mass proved to be a cirsoid neurofibroma, which involved the facial nerve from the midportion of the horizontal segment into the parotid gland. When the tumor was removed, there were five separate distal branches of the facial nerve. Nerve grafting was performed with an excellent result.

Maxwell has seen the facial nerve involved extensively by a plexiform neurofibroma in two other cases, boys of 1½ and 2 years of age, without any impairment of function. In each instance the operation was performed for removal of a tumor below the angle of the mandible, and apparently extending into the parotic gland.

Maxwell concludes that

> although there was no facial paralysis in the three cases of cirsoid neurofibroma of the facial nerve cited here, it would seem quite likely that facial paralysis would be produced if the tumor were to originate within the confines of the Fallopian canal, as in the reported cases of neurinomata. The gradual stretching of the nerve in the soft tissues seems not to produce paralysis. It does seem remarkable, however, that complete function of the facial muscles had been retained in the patient whose neurofibroma extended through the entire vertical course of the Fallopian canal.

Wade reported a case of a woman aged 52, in whom a facial palsy had developed gradually within two years.

A tumor, about 2 cm. in diameter, was felt in the deep lobe of the parotid gland, and an operation proved it to be a neurinoma, originating from the stem of the facial nerve just before the division.

Finally, O'Keefe reported a case of a tumor in the parotid region, the size of a lemon, in a man 57 years of age. It had developed gradually within two years, and for six months a facial palsy had been present.

It was removed, and proved to be a neurinoma.

Thus it will be seen, that in the cases described above it was impossible to arrive at the correct diagnosis preoperatively.

In Maxwell's cases there were not even traces of facial palsy. A diagnosis of a tumor of the parotid gland, or at the angle of the jaw extending into the parotid gland, was arrived at in all cases reported.

It should be stressed only that when a tumor of the facial nerve outside the stylomastoid foramen is removed, every effort should be made to bridge the gap between the stem of the facial nerve and the distal ramification. That this is possible has been demonstrated most excellently by Maxwell and Lathrop.

Tumors of the Surrounding Tissues.—Any tumor along the extratemporal course of the facial nerve may impair and eventually completely destroy facial nerve function.

The commonest tumors are those arising from the lymphatic glands at the angle of the jaw, and tumors of the parotid gland.

If the tumors are of a benign structure, it may be possible to spare the facial nerve during removal, and this applies especially to benign tumors of the parotid gland, because the branches of the nerve do not penetrate the substance of the gland but lie between the deep and the superficial lobe. If the nerve is accidentally injured during removal of the tumor, it should be repaired immediately.

If, however, the tumor is of malignant structure, attempts to save or repair the facial nerve are in the vast majority of cases doomed to failure, and plastic operations must be resorted to.

Summary

Tumors causing a peripheral facial palsy may originate either from the facial nerve itself, from the acoustic nerve, or from the surrounding tissues along the entire length of the facial nerve. A description of the pathology is given. According to the site of the tumor, the material may be divided into (1) intracranial, (2) intratemporal, and (3) extratemporal tumors.

Among the intracranial tumors the acoustic neurinomas and the epidermoid tumors are by far the commonest among those neoplasms which may cause a facial palsy.

The intratemporal tumors may arise either from the facial nerve itself or from the surrounding tissues. The principal tumors originating from the facial nerve are the neurinomas and the sarcomas.

A review is given of the 29 cases of neurinomas previously published, and another 3 cases are recorded.

Neurinomas may arise either from the vertical segment of the facial nerve (23 cases) or from the horizontal part (9 cases). The clinical picture is described, and it is concluded that Altmann's assumption that it is an easily recognizable clinical entity does not hold true any longer. A distinction, which is described in detail, must clinically be made between tumors arising from the vertical and from the horizontal segment. This distinction applies especially to the early stages of the neoplasms which it is so important to recognize in order to achieve good therapeutic results.

Sarcomas arising from the facial nerve are extremely rare, only two cases having been described.

The diagnoses and differential diagnoses are described in detail.

The prognosis quoad vitam is favorable. Some reservations would, however, be appropriate; thus a tumor near the middle ear and the labyrinth implies the risk of complications from the latter when the tumor grows into them.

Prognosis for future function of the facial nerve, on the other hand, is extremely bad, if the nerve is not repaired at the site of the lesion.

Treatment is exclusively surgical. The tumor should be removed as soon as the diagnosis is established. It is just as important to repair the facial nerve at the same session by nerve grafting.

Lastly, the extratemporal tumors which may cause a facial palsy are described. They, too, may originate from the facial nerve proper or from the surrounding tissues. They should, according to their nature, whenever possible be removed, and the facial nerve should be repaired.

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A Critical Study of a New Cerumenolytic Agent

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The medical literature contains many references [1-5] to the dangers of excessive or impacted cerumen, yet there is little to offer pharmaceutically to either physician or patient for its safe, convenient, and effective removal. Many of the preparations recommended for this purpose have been abandoned when it was found that they were not effective for the intended purpose, or, in some instances, introduced secondary problems which interfered with their efficacy. This report describes the development and study of a new cerumenolytic which has been found to be a safe and effective agent for the removal of excessive or impacted cerumen.

In 1947, Senturia and associates [6] reported that many formulations which claimed to be efficient cerumenolytic agents were, in effect, worthless. According to this group's findings, irrigation with distilled water and hydrogen peroxide remained the best means of disintegrating ear wax. However, since 1947, there have been many advances made in the pharmacy of topical medications [7-9] and the surface chemistry [10-11] of natural waxes. We felt it desirable to reevaluate the problem of cerumenolysis against the background of the newly developed scientific techniques for handling natural waxes and waxy exudates.

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The Characteristics and Occurrence of Cerumen

Cerumen consists of secretions of the ceruminous and sebaceous glands, dead desquamated epithelial cells, and bits of entrapped foreign matter. A recent analysis [12] of dried ear wax indicates the following composition:

Fatty acid	1.21%
Fats	17.67%
Cholesterol	3.54%
Cerotic acid	2.77%
Neurostearic acid	0.50%
Crystalline substance ($C_8H_{14}NO_2$—M. P. 204 C.)	1.17%
Protein	61.76%
Ash	4.93%
"Bitter substance"	trace

Senturia et al.[13] identified a number of amino acids in cerumen and also found that there was individual variation in lipid and protein content among the same age group—and even in the same individual, although no significant difference, based on age or sex, was observed.[14]

Carpendale,[15] who studied the ears of 1,000 healthy young men, reported his findings with regard to quantity and consistency of cerumen as follows:

	Normal	Pathological
Number of ears examined	1,790	210
% of ears containing wax	27.6	23.4
% of ears containing wax which totally obscures tympanic membrane	22.2	30.7
Consistency of wax		
Medium	90.5	—
Hard	5.1	—
Soft	4.4	—

Clinical Significance of Cerumen

At one time, there was an erroneous belief that wax from the ear was bactericidal, but some 30 years ago, Negus and Creed [16] showed that old wax forms a good culture medium for organisms. Not only may cerumen serve as a nutritive source for infecting organisms, but by increasing the pressure on the drum in an acute infection of the middle ear, it increases the pain and other symptoms of the disease. It may interfere with the drainage of the infectious material even after rupture of the drum and so increase the danger of complications.

Collins [17] suggests that impacted cerumen results when the secretion of the ceruminous glands is insufficient to liquefy the thick waxy secretion of the sebaceous glands. Others [16-19] believe that excessive secretion, anatomic abnormalities of the canal, excessive amount of hairs or foreign matter, or chronic inflammation may cause these plugs to form.

Itching, pain, sense of fullness, autophony, and deafness may result from impacted cerumen.[2,18] Frequently, there is a persistent dry cough from irritation of the tympanic branch of the vagus.[22] Occlusion may occur quite suddenly if water enters the canal and causes the wax to swell. Tinnitus and even vertigo may result because of pressure against the drum.[1-3]

Seltzer [4] reported that aural vertigo may occur while swimming, and this is usually the result of cold water entering into the ears which, under certain conditions, causes stimulation of the labyrinth. This investigator [4] reported that swimmers experiencing the greatest degree of vertigo were those with wax in the ears, especially those with wax in only one ear. Vertigo, under these conditions, is frequently accompanied by panic, leading to drowning.

Wax in one ear only, under other conditions besides swimming, may cause vertigo upon the entrance of cold air, instead of water, into the ear without wax. "It is this rather than the presence of wax itself which is the cause of vertigo." [4] Removal of the impacted cerumen is necessary to relieve these symptoms.

The prophylactic value of removal of hard ear wax was pointed out by Senturia.[20] Where there is an external otitis of any category, cerumen along with other debris should be removed whenever possible.[21-23] "Thorough cleansing of the canal reduces the amount of dead tissue, just as does good debridement of a contaminated wound." [21]

The Removal of Cerumen

The method of removing cerumen has been standardized: Syringing the ear, with minimal manipulation by means of a blunt-edged probe, remains the basic technique. While this method presents no problem to the otologist, there are certain inherent limitations in this procedure for the nonspecialist. It is usually advisable and necessary to soften the plug before it can be removed. Frequently, when the cerumen has been impacted over a long period of time, the outer layer of the epithelium of the canal becomes attached.[1] Instillation of an effective cerumenolytic agent is then indicated to prevent abrasions or tissue trauma which might result from the use of instruments or movement of the hard plug.

It was in this regard, as well as from the aspect of the utility of the agent as a vehicle, that in 1947 Senturia and Doubly [6] investigated the then available products. By means of an in-vitro test conducted on a "strictly qualitative basis," they [6] noted that many of the preparations suggested as cerumenolytics did not exhibit this property under their experimental conditions.

Other investigators, however, did not agree entirely with Senturia and Doubly. For instance, whereas the latter noted that the "oily preparations did not show any disintegrating effect," Hinchcliffe,[24] in a recent report of his findings regarding the efficacy of certain cerumenolytics, concluded that the data for olive oil were not applicable to other oily preparations, since an oily-base cerumenolytic was not found to be significantly effective in the same study.[24]

TABLE 1.—*Literature Comments on Suggested Cerumenolytics*

Agent	Literature Reference	Comments
A commercial mixture of sulfated vegetable oil, 25% liquid petrolatum, 50% water	Senturia[6]	Slow surface softening (In vitro); softens cerumen, water-miscible
	Little[23]	Effective when added to syringe water
A commercial oil-base product	Hinchcliffe[24]	Not significantly effective in helping to remove cerumen
Olive oil	Hinchcliffe[24]	Effective in helping to remove cerumen
	Little[23]	Not suitable because not miscible with water
Sodium carbonate solution	Hinchcliffe[24]	Not significantly effective
	Senturia[6]	Some disintegration (in vitro)
Hydrogen peroxide	Hinchcliffe[24]	Moderately effective, did not cause much discomfort
	Little[23]	Break up plugs, but may cause pain by causing swelling of plug; may cause dermatitis
	McLaurin[26]	Dissolves cerumen, crusts, scales; has no sensitizing properties; does not harm skin
	Fowler[21]	Softens wax
	Senturia[6]	Complete disintegration in vitro
Propylene glycol (vehicle for many ear solutions)	Leigh[29]	Excellent results in treating of otitis; no discomfort
	Senturia[6]	Caused swelling of cerumen (in vitro)
Glycerin	Little[23]	May cause dermatitis
	Brown[30]	"Patch tests have shown it to be free from irritating effects"; widely used
	Senturia[6]	Slow surface softening (in vitro)
Ethyl alcohol 95%	Senturia[6]	No action on cerumen (in vitro); good cleansing agent; evaporates quickly
Saline—isotonic or hypertonic	Senturia[6]	Rapid disintegration (in vitro)
	McLaurin[26]	Satisfactory
	Fowler[21]	Suitable for irrigation
	Leigh[29]	Suitable for irrigation

There appear to be other conflicting opinions about the different agents which have been recommended for use in the removal of cerumen.

A search of the literature has revealed many varied opinions as to the efficacy of claimed cerumenolytics (Table 1). It is obvious, therefore, that the problem of providing a satisfactory, effective, safe cerumenolytic agent remains to be solved.

Experimental

It was decided to approach the problem of cerumenolysis from the viewpoint of incorporating a surface-active agent with a viscous, nonvolatile, aqueous-miscible carrier. Propylene glycol was selected as the carrier because it most closely satisfies the criteria for this purpose. This agent is virtually nonsensitizing, and has been widely used in otologic and dermatologic practice. Propylene glycol is stable, constant in character, and without physiologic activity in the ear canal. It is completely miscible in water, and compatible with otologic medications.

The chemical analysis of cerumen made it evident that the surface-active agent of a cerumenolytic preparation would have to satisfy a special set of requirements not ordinarily encountered in lipid surface chemistry. The presence of a large quantity of protein material, which is polar in character, would have to be considered in attempting to attain the lowered interfacial tension system required for effective cerumenolysis.

The surface-active agent chosen is a special one * consisting of the fatty acid condensate of a protein polypeptide having the general formula: $C_{17}H_{23}CO.HN$ (CHR. $CO.NH)_nCHR.COOMe$ in which (CHR. CO.NH) denotes the amino acid radicals forming the polypeptide moiety, "n" stands for the number of amino acid radicals in the molecule, and "Me" indicates the salt-forming group, triethanolamine.

Examination of an aqueous solution of this surfactant with the ultramicroscope reveals the well-defined picture of a colloid.

* Cerapon, brand of triethanolamine polypeptide oleate-condensate.

The repeat pattern of the CO.NH groupings is the source of the protective colloid characteristics, which include strongly developed dispersing and emulsifying capacities.

A strong hydration potential originates from the CO.NH pattern which is also responsible for the high solubility of this agent. This surfactant lacks the aggressive tendencies and the substantivity of the strongly dissociated detergents and, consequently, does not produce dryness of the skin. By means of a buffering action, it protects the skin against alkaline attack, and has been reported, by virtue of its colloid nature, to afford protection against possible sensitizing agents without losing its own effective properties. It was felt that the polypeptide linkage of the surfactant would also provide a chelating substrate which would preferentially attack the protein composition of the cerumen. The polypeptide-fatty acid condensate, moreover, exhibits excellent surface-active properties and has been shown to be nonirrating and nonsensitizing to human skin [25]

The problem of producing an essentially water-free mixture of a surface-active agent and propylene glycol was accomplished through special manufacturing techniques. To this virtually anhydrous, viscous mixture was added 0.5% chlorobutanol as an antibacterial preservative to insure a sterile solution and to minimize the possibility of cross infection through accidental contamination. This preparation † was intensively studied for its safety and usefulness as a cerumenolytic agent. A summary of its physical and chemical properties is presented in Table 2.

† The cerumenolytic preparation of this investigation consists of Cerapon 10% in propylene glycol U. S. P. with 0.5% chlorobutanol U. S. P.; available as Cerumenex or Cerulan.

Rabbit Eye Irritation Test

Since surface-active agents have been shown to be irritating to the skin, it was important to determine the "irritation index." A sensitive and reproducible test, which quantitatively measures the irritating qualities of a substance by introducing it into the eyes of the albino rabbit, has been developed by the Laboratories of the United States Food and Drug Administration.[26] The degree of irritation and injury produced is observed over a period of seven days (or longer), and a preparation is considered to be satisfactory if the irritation produced has completely disappeared within a seven-day period. It should be remembered that this test is extremely sensitive, and only the most benign agents will produce little or no conjunctival reaction. Therefore, a substance which causes local conjunctival response lasting for a day or two, is considered essentially innocuous.

The results of this test (Table 3) indicate that this solution is essentially nonirritating and meets the critical limits for this test. The minimal ocular responses observed are of the same order as those of conventional otologic preparations in present-day clinical use. The new product does not introduce any new factor of acute local irritation. This degree of safety and lack of irritation is important in the use of the cerumenolytic in pediatric practice, where the possibility of accidental introduction into the eye may

TABLE 2.—*The Physical and Chemical Properties of Cerumenex*

Property	Cerumenex	Significance
Viscosity	50 centipoise at 25 C	Evenly maintained flow throughout ear canal
Surface tension	32.0 dynes/cm	Low surface tension insures wetting and penetration of ear wax
Hygroscopicity	High	Absorption of moisture present in transudates of ear canal
Aqueous miscibility	Complete	Ready mixing with physiologic fluids for even distribution Ease of removal by simple irrigation
pH	pH 5.6	pH of tissue surface of ear canal in normal state is on acid side
Volatility	Low vapor tension	Eliminates possibility of vapor pressure from droplets occluded by wax
Specific gravity	1.040 at 25 C	----
Odor	Odorless	Used in close proximity to organs of smell

TABLE 3.—*Results of Rabbit Eye Irritation Study with Cerumenex* *

	Day						
	1	2	3	4	5	6	7
Unwashed							
Rabbit 1	10	4	2	0	0	0	0
Rabbit 2	8	2	0	0	0	0	0
Rabbit 3	10	2	0	0	0	0	0
Washed after 2 seconds							
Rabbit 4	2	0	0	0	0	0	0
Rabbit 5	0	0	0	0	0	0	0
Rabbit 6	0	0	0	0	0	0	0
Washed after 4 seconds							
Rabbit 7	0	0	0	0	0	0	0
Rabbit 8	0	0	0	0	0	0	0
Rabbit 9	0	0	0	0	0	0	0

* According to the procedure of Draize, Woodard and Calvery: Methods for the Study of Irritation and Toxicity of Substances Applied Topically to the Skin and Mucous Membranes, J. Pharmacol. & Exper. Therap. 82:377, 1944.

occur, as well as to demonstrate its broader applicability to the use in the ear.

Local Tissue Effects

The possible local tissue effects arising from the use of the new cerumenolytic agent were investigated by studying the histology of the ear canal of rabbits after the instillation of the test compound and control solutions. The dosage and frequency of application adopted for this test were in excess of that which would normally be utilized in clinical practice in order to stringently assess the local effects of this agent. A paired-ear technique was adopted in which each rabbit received the test solution in the right ear and an identical control treatment of either water or a well-known glycerin-base otologic preparation ‡ in the left ear. Twenty albino rabbits, each weighing approximately 2.5 kg. and maintained on a colony diet, were observed for a preexperimental period of 14 days to assure normalcy. The rabbits were then divided into four groups:

Group I consisted of eight rabbits with intact tympani. Two milliliters of test solution was instilled into the right ear of each animal and two milliliters of either water or "glycerin-base preparation" was instilled into the left ear. The instilled material was allowed to remain in contact with the ear canal for 30 minutes, after which each ear was flushed with 5 ml. of tap water. This procedure was carried out three times a day for seven days.

Group II consisted of four rabbits with bilateral tympanotomy. The same test procedure was carried out as in the animals of Group I. All rabbits received the test solution in the right ear; two received the glycerin preparation, and two received the water in the left ear.

Group III consisted of four rabbits with intact ear drums treated as above except that the flushing procedure was omitted and the treatment period was limited to two days of three treatments per day.

Group IV consisted of four rabbits with bilateral tympanotomy treated exactly as Group III.

During the period of study, frequent gross examination of the external ear canal disclosed no changes which could be attributed to the action of the cerumenolytic agent per se. Animals from each group were killed at 7 days, 14 days, and 21 days after completion of the treatment schedule for Groups I and II, and all ears were examined § for gross and microhistologic changes.

The results of this study indicate (Figs. 1 and 2) that there were no tissue injuries arising from the use of this test solution. The histopathologic response observed after the use of the water or the glycerin base control solutions was virtually identical with that of the cerumenolytic agent. Although some degree of erythema and other reversible changes in the corium were present in both the control and the test ears of almost every rabbit, there were no signs of acute inflammatory reactions. The minimal tissue responses may have occurred as a result of mechanical irritation by the ear wax; trauma caused by the manipulative procedures, possible clinical infection, or by maceration due to the moisture occluded in the canal. Of special significance is the finding that there was no evidence of middle ear involvement in those rabbits with surgically punctured ear drums.

‡ The "glycerin-base" control preparation consisted of a glycerin solution of antipyrine and benzocaine.

§ The gross and microscopic examinations of the rabbit ear tissues were conducted under the supervision of J. C. McGrath, M. V. M., Philadelphia.

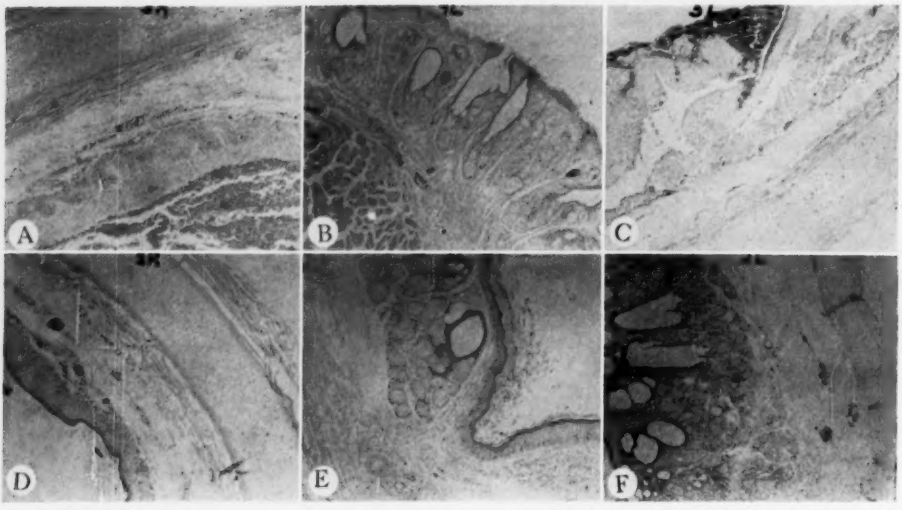
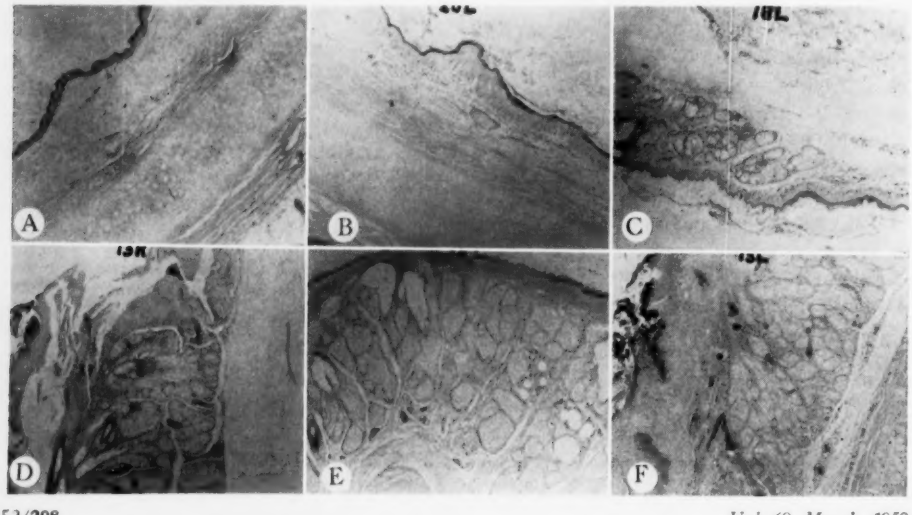


Fig. 1.—The comparative effect of Cerumenex on the histology of the rabbit ear. Cross section of the canal at junction of tympanic membrane; ×60; hematoxylin-eosin stain. Above, Group 1—ears flushed, drums intact, seven days after treatment: *A*, test ear; *B*, control ear—water; *C*, control ear—base preparation. Below, Group 2—ears flushed, drums punctured, 14 days after treatment: *D*, test ear; *E*, control ear—water; *F*, control ear—glycerin-base preparation.

The nonirritative nature of the new cerumenolytic agent is further established by the results observed in Groups III and IV, wherein the material was repeatedly instilled into the ear without flushing in order to maintain prolonged contact. The ears receiving the test solution, under these conditions, showed no difference in histologic

Fig. 2.—The comparative effect of Cerumenex on the histology of the rabbit ear. Cross section of the canal at junction of tympanic membrane; ×60; hematoxylin-eosin stain. Above, Group 3—ears not flushed, drums intact, 21 days after treatment: *A*, test ear; *B*, control ear—water; *C*, control ear—glycerin-base preparation. Below, Group 4—ears not flushed, drums punctured, seven days after treatment: *D*, test ear; *E*, control ear—water; *F*, control ear—glycerin-base preparation.



response from the ears receiving either of the two control solutions. Since the conditions imposed in these tests far exceed the dosage and frequency of use in clinical practice, it may be concluded that the local tissue safety of the new cerumenolytic agent is established.

Bacteriologic Study

While a cerumenolytic agent is not intended as an anti-infective, its germicidal properties are significant because of the possibility of cross contamination during its use. It is of interest to note that the presence of a bactericidal agent (or preservative) is recommended by the Food and Drug Administration [27] for ophthalmic solutions.

The antiseptic properties of the test solution were studied by the method of Rdzok et al.[28] This procedure is essentially a "challenge" technique in that it is designed to determine whether organisms, when added to a pharmaceutical preparation, will grow, remain static, or die. Three test organisms, which were considered typical and representative of those which might contaminate an otologic preparation, were used: a Coccus, Micrococcus pyogenes, var. aureus (ATCC 6538), a Gram-negative rod, Pseudomonas aeruginosa (ATCC 8689), and a fungus, Trichophyton interdigitale (ATCC 9533).

The results of the test (Table 4) indicate that the growth of the commonly encountered bacterial contaminants, cocci and Gram-negative rods, typified by a Micrococcus and a Pseudomonas, are efficiently suppressed by the antibacterial action of the solution. A good degree of bactericidal activity was present, since the number of bacteria per aliquot was reduced from 900,000 to 10 within one day. The fact that this minimal plate count was maintained throughout the seven-day test period further establishes the preservative action of the test solution. Fungistatic activity was also indicated, although this seems to have proceeded more slowly than the concomitant bactericidal action. This may be attributed to the more resistant sport form of this organism.

The results of this study establish that Cerumenex affords adequate protection against the various types of microbial contamination which might occur during its use.

TABLE 4.—*Preservative Efficacy of Cerumenex*

	Plate Count Days After Inoculation			
Organism	0	1	3	7
M. pyogenes var. aureus	900,000	10	10	10
P. aeruginosa	950,000	10	10	10
T. interdigitale	35,000	1,500	10	10

Sensitivity Screening

Although the components of the new cerumenolytic have been previously found to be nonsensitizing,[25] the preparation was screened for allergenic properties. The cerumenolytic agent was instilled into both ears of a group of 20 patients and allowed to remain for approximately 30 minutes, after which time the ears were swabbed. This procedure was repeated at weekly intervals for six consecutive weeks. A critical examination of the ears was conducted before each treatment. There was no evidence of either sensitization or local irritation observed after this intensive use of the cerumenolytic.

It is of interest to note that, in connection with sensitivity screening and allergenic properties, there was no instance of an allergenic response observed in the course of the broad clinical evaluation, in spite of the fact that this product was used in patients with known histories of allergy.

Clinical Study

The new cerumenolytic agent was investigated for its clinical efficacy in a series of 230 selected patients with varying degrees of excessive and impacted cerumen. The patient population represented a cross section of that encountered in everyday clinical practice and was obtained from the north, central, and southern regions of the United

States. Approximately 30% of the group were children (below the age of 16).

The presence or absence of aural pathology was determined by otoscopic examination, and the patients were classified (Table 5) into groups according to the observed amount of cerumen present. Group A consisted of those patients with impacted dry cerumen which obscured the eardrum. Group B consisted of those patients with excessive, dry, hard cerumen, but not obscuring the eardrum. Group C consisted of those patients with excessive amounts of soft ear wax.

The test material was introduced into one ear so that the ear canal would be filled, and the ear was plugged with cotton. After a short period (10 to 20 minutes) of contact time, both ears were gently flushed with water. The untreated ear served as a control to evaluate the degree of action induced by the flushing, per se. The ears were then examined with an otoscope and the results evaluated as follows: excellent, if complete removal had occurred; good, if "most" of the cerumen had been removed; and, poor, if little or no change occurred. The control ear was then treated with the cerumenolytic in the manner described above and the activity of the compound reappraised (Table 5).

Careful observation for any immediate and delayed tissue responses of the ear canal, were made, and follow-up examination was carried out (when feasible) within one week after treatment. The patients were also carefully questioned for the occurrence of any atypical subjective symptomatology which might be referable to the procedure.

In order to explore the cumulative effects of repeated instillation, a series of 20 patients were instructed in the use of this product and asked to repeat the procedure at biweekly intervals, on the day before returning for follow-up examination.

A dramatic cerumenolytic activity was observed in most instances, with complete removal of ear wax achieved in 204 patients (88.7%). A "good" degree of ear wax

TABLE 5.—*The Clinical Activity of Cerumenex*

Adults, Group *	Age Range	No. of Patients	Cerumenolytic Effect: Excellent †	Good ‡	Poor §
A	29-64 yr.	138	123	10	5
B	55-65 yr.	14	11	3	0
C	19-23 yr.	32	30	2	0
Total		184	164	15	5
Children, Group *					
A	3 mo.-16 yr.	28	24	4	0
B	9 mo.-16 yr.	7	7	0	0
C	4½ yr.-9 yr.	11	9	0	2
Total		46	40	4	2

* Group A—impacted dry cerumen, obscuring ear drum; Group B—excessive, dry, hard cerumen; Group C—excessive, soft cerumen.
† Complete removal of ear wax.
‡ Most of the ear wax removed.
§ Little or no change.

removal was obtained in 19 (or 8.2%) of the group, and the results were poor in the remaining 7 (3%). Approximately 6% of those patients in Group A (excessive, hard, dry cerumen, obscuring the eardrum) required at least two repetitive instillations to remove the impacted wax. These were evaluated by the same criteria as above, since the total contact time of the cerumenolytic and the accumulated wax was within the experimental test limits.

There were no instances of any untoward tissue responses observed in any of the groups. None of the patients reported any subjective symptomatology and most of them expressed enthusiasm with the results obtained. Concomitant aural pathology, when present, was not aggravated. However, there were too few instances of associated pathology to warrant the observation of a positive effect of ear wax removal on the course of the preexisting disease. Similarly, multiple instillation of the agent into the ear canal did not induce any adverse effects. The use of the preparation at either weekly or biweekly intervals did not result in any untoward tissue manifestations.

While the effects of the new preparation were experimentally determined after an

arbitrary contact period, it is recognized that the accepted and widely utilized clinical procedure is to instill a cerumenolytic at least one or more days prior to either irrigation or manipulation by the clinician. We concur with this practice, especially since the new cerumenolytic has exhibited a complete spectrum of lack of irritating or sensitizing properties.

The ease with which this agent facilitates the removal of ear wax is a striking contrast to the cumbersome operative irrigation techniques. This preparation is a safe, nonirritating, nonsensitizing cerumenolytic agent which effectively and conveniently facilitates the removal of ear wax. It has a broad range of efficiency which is most desirable in clinical otologic practice.

Summary and Conclusions

The current status of the problem of cerumenolysis has been reviewed, and an agent, Cerumenex, designed to meet the physico-chemical, as well as the physiologic requisites for satisfactory cerumenolysis has been investigated.

The new cerumenolytic was found to be nonirritating by the Draize-Woodard-Calvery rabbit eye irritation test. The local tissue responses to this drug were studied by repeated instillation into the ears of rabbits, and the responses obtained indicated an absence of any local histopathologic effects. Similar tissue changes occurred in virtually all of the test and control ears, and were of a short transitory nature. The agent exhibited a bacteriostatic and fungistatic effect when tested against Micrococcus pyogenes var. aureus, Pseudomonas aeruginosa, and Trichophyton interdigitale. The obvious advantage of maintaining a high degree of germicidal activity in a preparation which is intended to come in contact with affected areas, is satisfactorily met by this product.

A preliminary screening for allergenic reactions in 20 patients indicated the absence of either allergenic or sensitizing properties for this preparation. There were no allergenic reactions observed in the subsequent clinical trial of 230 patients, in spite of the fact that patients with known history of allergy were included.

The cerumenolytic efficiency, studied in 230 patients ranging in age from 3 months to 65 years with varying degrees of cerumen accumulation, indicated a high order of activity, which produced excellent results in 204, good results in 19, and poor results in 7.

Cerumenex, the new cerumenolytic agent, offers a safe, efficient and convenient means for the removal of ear wax. It is hoped that the wide use of this agent may replace the time-consuming, cumbersome, and sometimes traumatizing methods for the removal of cerumen from the external auditory canal.

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The Routine Use of the Minimal Caloric Test as an Office Procedure

Report of Experiences with One Hundred Patients

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An office test of labyrinthine function is not a quantitative test, but rather a qualitative one. The method to be described herein has several advantages, especially its simplicity and ease of operation. The technique to be described is not original, but has minor differences from other methods. The minimal caloric test has been reported upon by Veits (as quoted by Arslan [2]), Török,[9] and the University of Chicago Otolaryngology Department.

The technique of the examination is as follows: First the Frenzel spectacles are applied over the patient's eyes. The spectacles have 20+ D. lenses, which magnify the eyes so that any movement can be easily seen. The patient is examined for spontaneous nystagmus. The patient is asked to look to the right and then to the left; this evokes less fixation nystagmus than when the patient is asked to look sideways at your finger. Then 5 cc. of tap water at 80 F is injected against the posterior superior quadrant of the tympanic membrane through an ear speculum with use of a syringe and small metal cannula. The water is inserted over a period of three to five seconds. Time is counted from the moment the last drop of water is inserted. The latent period is measured by noting the time of onset of nystagmus. The duration is counted from the moment the last drop of water is inserted until nystagmus ceases. The patient looks straight ahead with his head bent downward at 30 degrees to the horizontal. The patient is told that he will not see clearly and that the purpose of the glasses is to magnify his eyes. We have not used the light incorporated in the Frenzel spectacles, but reflect a beam of light by a head mirror into the patient's eyes, at intervals.

In some patients a to-and-fro motion of the eyes develops due to visual disturbances induced by the Frenzel glasses. True nystagmus must be differentiated from this undulation.

The latent period is measured first. If no nystagmus appears in 35 seconds, turn the patient's head so that he faces up toward the ceiling. Often when there is no response from the vertical canals there will be a response from the horizontal canals. After the nystagmus is observed we measure the duration of the nystagmus and we count the number of nystagmus jerks. A count below 30 would be considered hypoactive. We also measure the amplitude of the nystagmus. Coarse amplitude would be larger than 3 mm. but is rarely elicited by this method. Medium excursions are 1 to 3 mm. wide. Small amplitude is less than 1 mm. wide. If the amplitude is just barely perceptible, I list it as tiny. The frequency in a 10-second period is counted. The usual frequency is from 6 to 18 jerks per 10 seconds. The end-point is taken as the point where regular nystagmus ceases.

After completion of the test the ear canal is wiped dry. As with any vestibular test it is preferable to wait five minutes before testing the other ear. If no response was obtained from the first ear, the other ear may be tested immediately.

If you wish to test for directional preponderance, 20 cc. of water at 115 F may

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be used as a stimulus. This stimulus is not as strong as the cool water and more water is needed.

A response is considered hypoactive if there is no response in the face-front position and the duration in the face-up position is less than 60 seconds. Usually in a hypoactive response the amplitude will be small and the frequency will be 6 jerks or less in a 10-second period.

Indications for Testing

The indications for vestibular examination may be listed as follows:

1. *Vertigo.*—In a case of endolymphatic hydrops the caloric test shows hypoactivity in the pathologic ear in about 70% of cases.[8] A case of vestibular neuronitis usually has a hypoactive result from the caloric test in one or both ears. Many cases of dizziness have a normal caloric reponse.

2. *Intracranial Lesions.*—In the past the principal application for the caloric test has been in neurologic or intracranial problems. However, these constitute only a small proportion of the cases where caloric tests are applicable, probably less than 10%. The incidence of cerebellopontine-angle tumors or other intracranial conditions in an otolaryngologist's practice is small. Most cerebellopontine-angle tumors produce a dead labyrinth. If there is no facial palsy, no head pain, normal corneal sensation along with an active labyrinth, the possibility of a cerebellopontine-angle tumor can be largely ignored.

3. *Perceptive Deafness.*—In cases with mild perceptive deafness the minimal caloric test is usually normal. In many cases of severe perceptive deafness the caloric reaction is hypoactive or absent. Unilateral perceptive deafness is an absolute indication for a caloric test. It is this sign which cerebellopontine-angle lesions exhibit.

Conductive deafness is usually not an indication for caloric testing. Some men advocate a caloric test prior to a fenestration operation to pick up rare cases of absence of the horizontal semicircular canal. A unilateral perceptive deafness may be mistaken for a unilateral mixed deafness due to borrowed bone conduction from the good ear. A caloric test in unilateral conductive deafness may be of value.

Stoner[8] studied the results of caloric testing in deafness. He used the Hallpike directional preponderance test. He found normal responses in acoustic trauma; 73% of his series of congenital or perceptive deaf cases gave a normal response. Arnvig[1] found that the great majority of deaf children with inherited hearing loss had normal vestibular function. Abolished vestibular function in congenitally deaf children should arouse a suspicion of retinitis pigmentosa. Vestibular function is abolished in most cases of severe acquired hearing loss in children. Arnvig used the standard Bárány test.

4. *Chronic Middle-Ear Infections.* — I usually use a Dundas Grant cold-air test with Frenzel glasses. Water may cause a flare-up of middle-ear infection. A hypoactive or absent reaction indicates that the labyrinth is affected and helps to define the necessity for a mastoidectomy.

Results of Minimal Caloric Test in One Hundred Patients

Indication	No. of Persons	Results of Minimal Test		
		Normal	Hypoactive	Absent
Dizziness				
(a) Endolymphatic hydrops	6		5	1
(b) Vestibular neuronitis	5		1	4
(c) Positional vertigo	5	5		
(d) Other cases of dizziness	8	7	1	
Perceptive deafness				
(a) Severe bilateral	8		2	6
Severe unilateral	4	3		1
(b) Moderate	35	31	4	
Otosclerosis	4	4		
Adhesive otitis media	2		2	
Perforated tympanic membrane	8	8 (Hyperactive		
Head trauma	3		1	2
One ear				
Complaints	12	10	2	

5. *Head Trauma.*—Head trauma can cause a hypoexcitable vestibular labyrinth by peripheral effect or by the effect on the nuclei in the brain stem. Testing is particularly important in medicolegal cases where malingering may be a factor. A vestibular test furnishes objective evidence of damage.

Results in a Series of 100 Tests in the Office

All the reported cases are adults (Table). A total of 24 patients were tested for the complaint of dizziness. Six of these met the strict audiologic criteria for endolymphatic hydrops and all six gave a hypoactive response to the caloric test. Five cases were diagnosed as vestibular neuronitis or toxic labyrinthitis. These five cases showed a diminished reaction to the minimal caloric test in both ears. There were five positional vertigo cases who gave a normal reaction to the minimal caloric test. Most of the remainder of the dizziness cases gave normal reactions to the minimal caloric test.

In this short series there was only one patient suspected to have an intracranial lesion. He had severe unilateral perceptive deafness with absence of any response of this ear to even mass caloric test. He had some dizziness. He refused any further investigation. Review a year later revealed no further change.

There were eight cases with severe bilateral perceptive deafness where the audiograms revealed a loss of 75 db. or worse. All gave a markedly hypoactive or no response to a minimal caloric test. Three cases of severe unilateral perceptive deafness with tinnitus had normal reactions to the minimal caloric test. Thirty-nine cases of moderate perceptive deafness or conductive deafness, including acoustic trauma, presbyacusis, and otosclerosis, revealed normal or only slightly hypoactive responses. The minimal caloric test was not helpful in these cases.

In this series there was no case of middle-ear or mastoid infection with an absent caloric reaction. Hyperactive reactions were elicited in cases with perforated tympanic membranes or cases with radical mastoid cavities. This is to be expected as the stimulus is closer to the labyrinth and its effect stronger than ordinary. Two patients with badly scarred tympanic membranes had hypoactive reactions.

Only three patients are included with head trauma. Two had a unilateral dead labyrinth with no hearing and no caloric response on the affected side. One case had a normal audiogram and a moderately hypoactive response on the affected side.

The remainder of the series had vague ear complaints such as ear pains and stuffiness. Two of these were nervous women who developed a hyperactive response with dizziness to the minimal caloric test. The others gave normal responses, except for two with hypoactive responses.

Advantages of This Test

The chief advantage of the minimum caloric test is the lack of disturbance to the patient. The test rarely produces any vertigo and the temperature of the water is not disturbing.

A second advantage is that, even though the test is not quantitative, it seems to reveal hypoactivity better than other office tests.

The test is rapid. Only brief preliminary explanations are necessary.

Disadvantages

In doubtful vertigo cases it is an advantage to produce a standard rotary vertigo with which the patient may compare his own dizziness.

Young children will not cooperate. They will not hold their eyelids open so that you can see the nystagmus through the Frenzel glasses.

Where the test gives no response, it is necessary to do a mass caloric test.

Summary and Conclusions

A description is given of the minimal caloric technique for testing the function of the semicircular canals. The advantages and disadvantages of this technique are de-

scribed. Experiences with this test in vertigo, deafness, intracranial infection, and head trauma are given. A report of our experiences with 100 patients tested in the office is given.

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Combined Neurofibroma and Carcinoma in the Larynx

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Of the benign neoplasms which appear in the larynx, neurofibromas are usually considered to be one of the rarest. In the past they have been reported occasionally either as isolated tumors confined to the larynx [1-11] or accompanying multiple neurofibromatosis.[12] Their rarity is attested to by the report of New and Erich,[13] who found one such neoplasm in a review of 722 cases with benign lesions in the larynx seen at the Mayo Clinic over a 30-year period. Van Loon and Diamond [14] also reported only one laryngeal neurofibroma during a 12-year period in which a large group of laryngeal lesions were seen. Fisher and Odess [15] found 19 cases reported and added 2 more cases. Figi and Stark [16] in a later study, observed that 25 cases with laryngeal neurofibromatosis had been reported in the world literature by 19 authors and added 5 similar cases.

The neurofibroma is a nonmalignant new growth of neuroectodermal origin. It is usually associated with peripheral nerves, but may appear in nerves of the central or autonomic nervous systems. This tumor is typified by diffuse proliferation of the peripheral nerve elements. It may appear as an isolated tumor or more commonly as von Recklinghausen's disease (multiple neurofibromatosis), a disease which has a strong hereditary tendency. Commonly neurofibromas appear at the end of a nerve, often in the skin, producing small nonencapsulated nodules which may have pigmentation in the overlying skin. The neurofibroma may also appear as a diffuse thickening of the skin, termed elephantiasis neuromatosa. Another form of neurofibroma appears inside nerve sheaths. In plexiform neurofibromas the sheaths become thickened and tortuous. This process may progress along a nerve until the spinal cord is reached and involved. Another finding often seen in von Recklinghausen's disease is the pale-brown, variable-size, patchy pigmentation in the skin, called café au lait spots. These are caused by melanin which is found in the deeper layers of the epidermis. A less common form of multiple neurofibromatosis is the fibroma molluscum. This is caused by a proliferation of the fibrous tissue in the outer layers of the corium. The result is a raised and often pedunculated mass. The histological picture presented in neurofibromatosis is that of an irregular, diffuse proliferation of the Schwann cells. This may be accompanied by neurons which proliferate in a disorderly fashion, sometimes with the formation of Meissner corpuscles. Verocay bodies are not formed.

Available information indicates that in the larynx these tumors are found more frequently in women (3:2—Figi). They may appear at almost any age (from 6 years to 52 years—Figi), and may be variable in duration, usually quite slow growing (Van Loon). Their location in the larynx has varied, there apparently being no site of predilection. The size of the tumors has also shown considerable variation, in several instances the respiratory distress or hoarseness therefrom being the presenting symptom. Complete surgical removal of the lesions has met with a high degree of success. Although previous investigators had reported that these lesions more fre-

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quently are smooth, benign, encapsulated in appearance, Figi observed that they may appear in three forms: (1) smooth, discrete, encapsulated tumors; (2) diffuse, poorly localized, and nodular; (3) polypoid tumors on the vocal cord.

Patients with multiple benign new growths in various parts of the body are seen quite frequently. Also patients with multiple malignant neoplasms located in the same or in different areas, either of the same cell type or of cell types which are different histologically, are reported occasionally. In a previous paper [17] one patient was reported with a malignant neoplasm located in the esophagus, containing apparent squamous-cell carcinoma, adenocarcinoma, basal-cell carcinoma, and fibrosarcoma elements. Another patient developed four independent squamous-cell carcinomas, three in different locations in the oral cavity and pharynx, and the fourth in the right lower lobe of the lung.[18] However, in each instance of the latter case, the neoplasms were malignant forms of the same parent cell.

In the larynx, as elsewhere, benign new growths may appear. These include papillomas, fibromas, adenomas, angiomas, myomas, myxomas, chondromas, neurofibromas, and the borderline, primarily inflammatory, tumors which include vocal polyps, vocal nodules, hematomas and various cysts. Each of these tumors has its potential malignant counterpart. Usually the malignant version histologically resembles the parent cell type and is named from it.

Potentially premalignant lesions frequently seen in the larynx include hyperkeratosis and leukoplakia. In the event of malignant change these lesions usually degenerate into squamous-cell carcinoma. Occasionally adenocarcinoma is seen, and rarely basal-cell changes are observed. Usually the malignant tumor is of the same cell type and progresses from a parent lesion, which may be an alteration in the normal component parts of the larynx, or from a benign new growth which undergoes malignant change.

Mixed tumors containing more than one benign or malignant cell type are not often seen in the larynx. Mixed type tumors, containing one cell type which is malignant and one cell type which is benign, are infrequent. This is especially true where the malignant cell type is ectodermal in origin superimposed upon a benign tumor whose cell type is mesodermal.

Recently a patient was seen in whom generalized neurofibromatosis (von Recklinghausen's disease) had existed for many years. Hoarseness and increasing dyspnea brought him to laryngeal examination. Histological study revealed both epidermoid carcinoma and neurofibroma tissue in the sections from the larynx. Combined neurofibroma and squamous-cell carcinoma in the larynx has not been reported. This case is considered worthy of documentation.

Report of a Case

A 56-year-old white man complained of progressive hoarseness for six months and increasing

Fig. 1.—Photograph of the patient's head and neck showing the many cutaneous nodules.

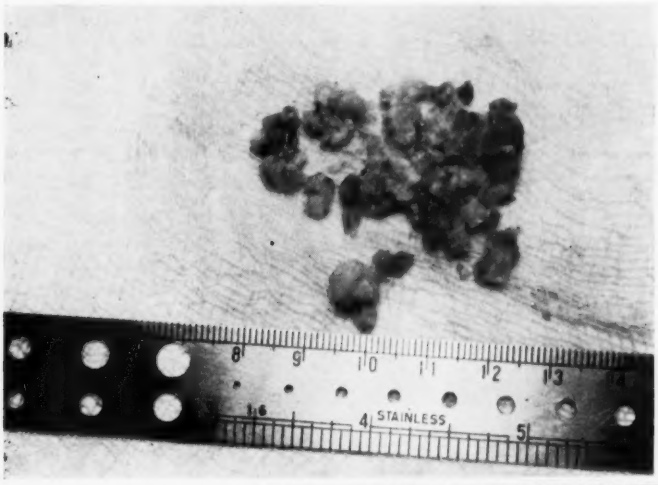

Fig. 2.—Tissue removed from the larynx by direct laryngoscopy.

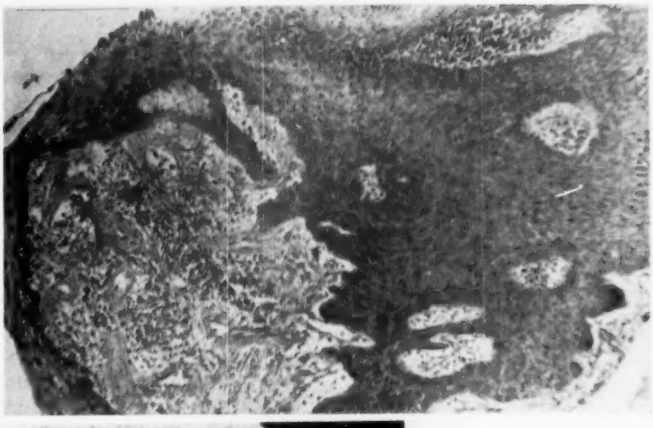

Fig. 3.—Photomicrograph of a section showing a nest of well-differentiated squamous-cell carcinoma.

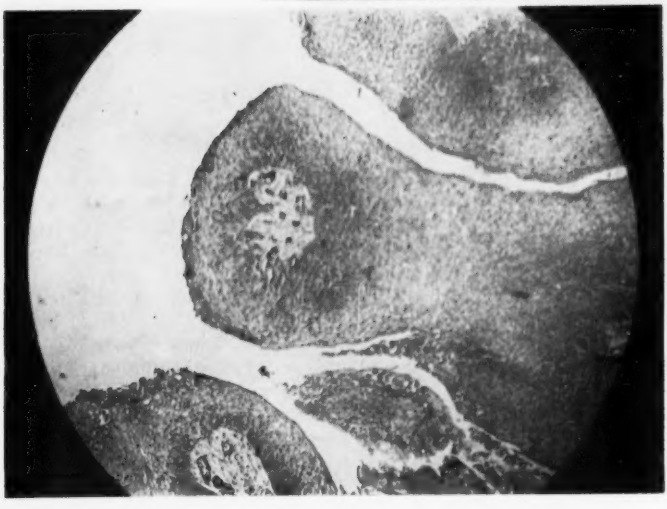

Fig. 4.—The papillary configuration of the superficial portion of the laryngeal tumor is well illustrated in this photomicrograph.

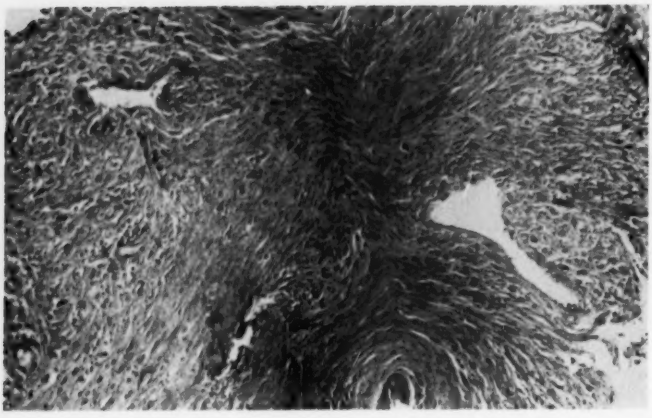

Fig. 5.—A photomicrograph of tissue removed from the basal portion of the laryngeal neoplasm. Note the diffuse, haphazard proliferation of the constituent components, the Schwann cells. No Verocay bodies are present.

dyspnea for one month. Previously he had had periodic hoarseness with colds and a "husky voice" for several years. Generalized variable-sized subcutaneous nodules and skin pigmentation resembling café au lait spots of multiple neurofibromatosis had been present for many years (Fig. 1). Except for the skin lesions, general physical examination was normal. A chest x-ray showed multiple, rounded, nodular lesions in both lung fields. There was no cervical lymphadenopathy.

Direct laryngeal examination showed a large, granular, multilobulated, friable, vascular, papillary mass arising from the mucosa of the anterior portion of the left true vocal cord on the superior, vocal, and inferior surfaces. The mass extended onto the anterior commissure and caused about 75% obstruction to the airway. Motion of the left cord was restricted but not absent. The tumor was removed entirely for biopsy (Fig. 2). Bronchoscopy was negative for endobronchial lesion.

Microscopically, the excised specimen showed a well-differentiated squamous-cell carcinoma (Fig. 3), exophytic type, with a definite papillary configuration (Fig. 4). In the tissue removed from the basilar portion of the papillomatous mass there was the histological pattern compatible with neurofibroma (Fig. 5).

X-ray therapy was administered to the laryngeal area with excellent response by the neoplastic process. Follow-up examination has shown no evidence of recurrence or metastasis in over a year. The patient has remained asymptomatic otherwise in this interval.

Summary

A résumé of the literature pertaining to the subject of neurofibroma in the larynx has been presented. This is one of the uncommoner new growths in the larynx. A case has been reported in which a man with long-standing von Recklinghausen's disease developed a neoplasm on the left vocal cord. Histological study of the tumor, after careful, complete local excision for biopsy, showed it to be primarily an exophytic, papillary type squamous-cell carcinoma. In the basal portion of the neoplasm there was the microscopic pattern suggesting neurofibroma. It would appear that this patient may have had a small neuroectodermal lesion on the left vocal cord for some time. The epithelium overlying the benign tumor apparently underwent malignant degeneration which resulted in an epidermoid carcinoma. Local excision followed by x-ray irradiation has apparently arrested the malignant portion of the tumor.

14 Riverview Heights.

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Aberrant Parathyroid and Thymus in the Pharynx

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The unusual presence of aberrant parathyroid and thymus tissue found in a child's pharynx recently at the Children's Hospital aroused our interest in the possibility of similiar cases and the circumstances in which they were found.

Embryologically the parathyroid glands arise from the dorsal branchial pouches III and IV. In the development of the embryo the parathyroid III moves more caudad than parathyroid IV, and both are usually found at the level of the thyroid gland at the latter's final position in the neck. The thymus develops from the ventral portion of the pouches III and IV, and in a similiar manner migrates to the superior mediastinum during fetal life.

Aberrant thymus tissue has been reported a number of times in the literature, its most frequent ectopic site being in the neck. Gilmour [1] describes 13 cases of aberrant thymus tissue he had found in the neck, usually in intimate relation with the carotid vessels. Description of aberrant thymus tissue in the hypopharynx has been reported by Epstein and Loeb [2] in a 3-week-old infant, but otherwise it has been difficult to find reports of thymus tissue present in the pharynx.

Aberrant parathyroid tissue has also been described, but much less frequently than thymus tissue. Gilmour described several cases in which parathyroid tissue was found at different levels in the neck. He describes one case in which a 12-week-old girl at autopsy had a right parathyroid in the submucosa of the hypopharynx at the level of the cricoid with a persistent ductus pharyngobrachialis running from the gland to the mucosal surface. In this case also, the thymus rudiments were found at the level of the internal carotid. Another case was reported by Askanazy (1911) of an accessory parathyroid attached to the phrenic nerve and found at autopsy. Baker and Brines [3] described a hyperfunctioning adenoma of an ectopic parathyroid gland which was situated in the lower neck lateral to the thyroid gland in an 18-year-old girl. A careful review of the literature failed to reveal any additional aberrant parathyroid case reports.

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Report of a Case

This was the fifth Children's Medical Center admission of a 10-year-old girl whose history dates from the time of birth, when multiple congenital anomalies were detected. She was admitted on the day of birth, and the following deformities were noted: imperforate anus, rectovaginal fistula, microtia left ear, left facial paresis, congenital heart disease with persistent right aortic arch posterior to esophagus, and left branchiogenic sinus. On that first admission she had a successful anoplasty and closure of the rectovaginal fistula, and at the age of 4 years she had an excision of the left branchiogenic sinus.

At the age of 9 years she began to complain of a lump in her throat and some dysphagia. Although it was known that she had a vascular ring compressing the esophagus, this had been asymptomatic. Examination of the pharynx revealed an absent left posterior tonsillar pillar and left palatopharyngeus muscle. The left tonsil appeared poorly demarcated, and continuous with it was a smooth pink 2 cm.×1 cm. swelling on the posterolateral pharyngeal wall covered with mucous membrane. The mass extended down to the left aryepiglottic fold. The right tonsil appeared normal and both pillars were present (Fig. 1). Neck examination revealed a well-healed 2 cm. scar at the angle of the mandible on the left side.

At the time of this examination, it was the impression that this mass might be a residual

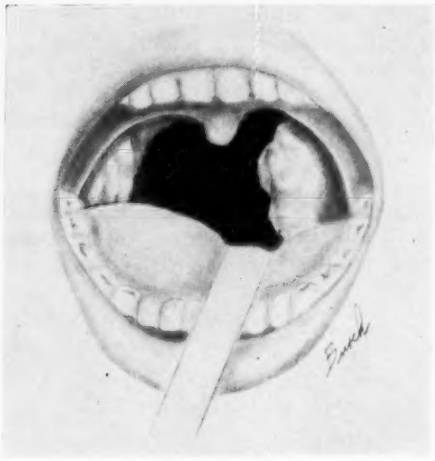

Figure 1

branchial cyst communicating with the pharynx. She was admitted to the hospital, and, under general anesthesia, the two masses in the left pharynx were excised. The larger mass seemed to have the consistency of a tonsil. The smaller mass felt fatty. The right tonsil, which appeared normal, was also removed at this time. The patient made an uneventful recovery.

The pathological report revealed the right tonsil to be normal in histological appearance. The larger mass taken from the left tonsillar region showed histological features similar to those of the right tonsil. The smaller mass, however, was described as having stratified squamous epithelium on the surface, but deep to this was a definite lobular arrangement of small dark lymphocytic cells with numerous Hassall's corpuscles within each lobule. Adjacent to this tissue was an additional structure composed of large polyhedral pale cells with dark central nuceoli (Fig. 2). The final diagnosis was left tonsil containing parathyroid and thyroid-thymic elements, and a portion of a branchial-cleft sinus.

Examination of the patient three months postoperatively failed to reveal any residual mass in the pharynx or hypopharynx, and there was no clinical or laboratory evidence of hypoparathyroidism.

Summary

A review of the literature on aberrant parathyroid tissue and thymus tissue is pre-

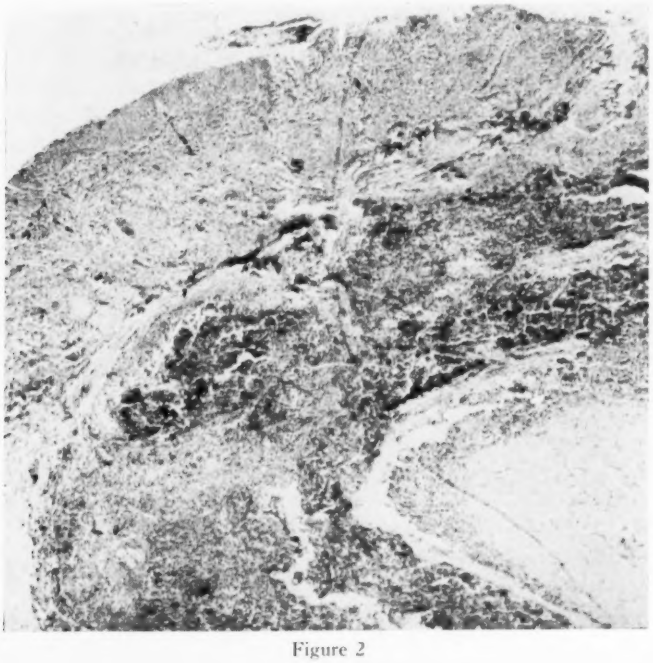

Figure 2

sented and illustrates the paucity of reported cases involving the pharynx.

A case report of ectopic parathyroid and thymus tissue simulating a tonsil in a 10-year-old girl is presented. No similar case could be found in the literature of a pharygneal aberrant parathyroid.

206 Waterman St. (6).

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

Clinical Application of Paired Masking Enclosures in Pure-Tone Air- and Bone-Conduction Testing

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Problems in Masking and Current Methods of Handling Them

Although the need for masking in pure-tone audiometric testing is generally recognized by workers in the field of audiology, a satisfactory procedure and device which can be used uniformly as a masking agent for valid and reliable testing is still unavailable. The variety of masking devices included in commercially available audiometers and the differences in the way they are calibrated, the lack of agreement among specialists engaged in hearing measurement of when and how much masking should be employed, particularly in the testing of bone conduction, and the differences in procedures for applying the masking device used by different testers attests to the general confusion which surrounds this enormously complex problem.

A New York audiologist has referred to masking as "the messiest problem in audiology." [1] Lüscher and Zwislocki say that "the theoretical basis of masking has not yet been worked out sufficiently." [2] Most specialists in audiology and related fields discuss the subject in cautious and guarded terms, refusing to commit themselves on specific aspects of the problem. This is because we are still unable to solve a number of important clinical problems involving the use of masking in spite of the prolific and important writings on the subject. Consequently, confusion exists regarding effective procedures and devices which can be employed in audiometric testing and there is little uniformity or standardization of practices in places where audiometric tests are conducted.

The use of effective masking techniques is of particular importance in testing the poorer ear of patients having a considerable difference in the sensitivity of the two ears (greater than 30 db.). When tones are presented to the poorer ear at intensities sufficiently strong to be audible to the patient, they are also strong enough to traverse the 21 cm. of skull which separates the two ears. These tones may then be heard in the contralateral ear, resulting in what is usually referred to as a "shadow curve." A spurious normal bone line on the poorer ear of a monaurally deaf patient which actually represents a shadow curve of the better ear may result in an incorrect diagnosis of the patient's hearing loss. Unless a bone-conduction test using masking or other special tests are performed to establish the true nature of the loss, the

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The research reported in the present paper was conducted at the Audiology and Speech Center of Walter Reed Army Medical Center, Washington, D. C., under the direction of Dr. James P. Albrite, Director, Dr. R. Edwin Shutts, Assistant Director, and Dr. Robert A. Butler, Supervisor of Psycho-Acoustic Research. The paper is based on a portion of a doctoral dissertation completed at Columbia University, New York City, under the direction of Dr. E. P. Fowler Jr., Dr. Moe Bergman, and Dr. Paul Lindenberg.

possibility exists that inappropriate treatment of the condition may be instituted. The question of how much masking should be used in this type of case is of particular significance because if insufficient masking is used on the better ear the results of testing the poorer ear will only show how well the normal ear is receiving across the skull sounds presented to the impaired ear. If, on the other hand, too much masking is used there may be a threshold shift on the poorer ear and greater amounts of masking may even obliterate the threshold of the poorer ear completely. The effective masking level for a particular patient differs from frequency to frequency, and a standard, "across the board" level which is used throughout the frequency range tested is subject to serious error in many cases.

Among the techniques which are employed to determine the type of loss present in an impaired ear are the direct tests of recruitment, difference limen tests, and speech discrimination tests. The most commonly used basis for differential diagnosis between middle ear and sensorineural deafness, however, is still a comparison of the air and bone lines on the impaired ear or ears of the patient, sometimes referred to as the "air-bone gap." While the *reliability* of bone conduction measurements using an audiometer which has been properly calibrated and a small vibrator of the type used with portable bone-conduction hearing aids is as high as that for air-conduction tests,[3] the *validity* of bone-conduction testing has not been established. That is, we do not know just what is happening when we apply the bone-conduction oscillator to the mastoid process of the patient's temporal bone. Studies on compression and inertia bone conduction by E. Bárány[4] and Békésy[5] suggest that we stimulate the cochleas of both ears regardless of where on the skull the vibrator is placed. Hirsh[6] has stated that it is almost inconceivable that we can stimulate one cochlea and not the other when a vibration is applied to the skull. Furthermore, the mastoid process may not be the best place to apply the bone-conduction oscillator, and an area located on the forehead is considered to have the greatest homogeneity of tissue by some writers.[7] This aspect of the problem is worthy of further research but will not be investigated in the present study where the conventional position will be used for bone-conduction measurements.

One important aspect of bone-conduction measurements is the problem of how much, if any, masking should be used routinely in bone-conduction testing. We do not know the final answer to this problem. At one audiology clinic in New York City masking of the contralateral ear is uniformly used in performing a bone-conduction test regardless of whether there is a marked differential between the hearing sensitivity of the two ears or whether the patient reports a lateralization of the tones to the opposite ear. This is based on the very real possibility that a tone fed into a bone-conduction oscillator placed on the mastoid process of one ear may stimulate the cochlea of the opposite ear as much as (or even more than) the cochlea of the ear under test. At another center in this area, masking is not used for bone-conduction testing unless there is a considerable differential (more than 30 db.) between the air conduction of the two ears and the patient reports a lateralization of the bone-conduction sounds to the opposite ear. The rationale of this approach is based on the possibility, also not without logical basis, that introducing a masking noise unnecessarily into the contralateral ear may cause a shift in the bone-conduction threshold of the ear under test. Furthermore, covering the nontested ear with a small enclosure, e. g., an earphone, in order to introduce a masking noise causes a "hypersensitization" of this masked ear, and tones presented to the tested ear will be lateralized to the ear covered by the earphone. This "Weber-like" phenomenon in bone-conduction measurement has been investigated in two Master's theses conducted at the University of Oklahoma under the direction of

Dr. John W. Keys.[8,9] One of these investigators [8] found that of 250 threshold measurements recorded in order to determine the lateralized threshold for each of 50 normal ears at each of the five test frequencies (250, 500, 1,000, 2,000, and 4,000 cycles) only 91 thresholds did not lateralize when a masking receiver was placed on the ear not under test. Such evidence appears to support the practice of masking only when "absolutely necessary," since the presence of an earphone over the masked ear clearly encourages a lateralization of the test tone to the nonexamined ear.

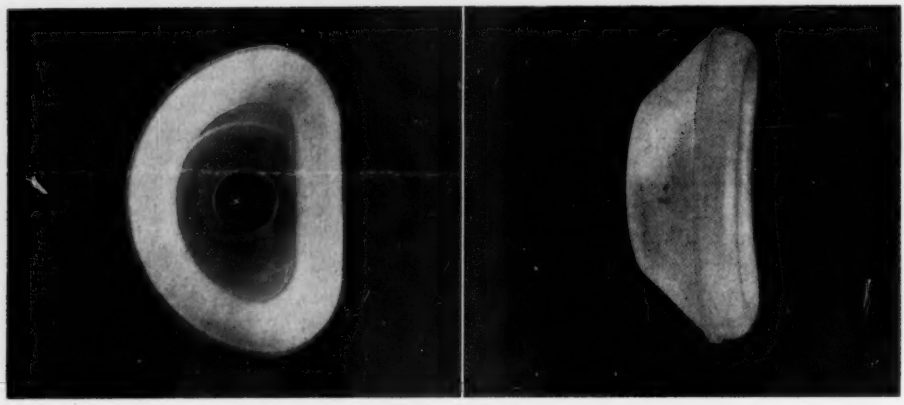
At still a third center, the Weber test with the bone-conduction oscillator of the audiometer is used to determine which ear should be masked in bone-conduction measurements.[10] The Weber test is performed at those frequencies which will be tested by bone conduction. The lateralized ear on the Weber test is assumed to be the ear with the better bone conduction and is tested first without masking. The bone conduction of the nonlateralized ear is then tested with masking presented to the lateralized side. Since lateralization on the Weber test may change as a function of intensity, particularly when recruitment is present, a level of 5 or 10 db. above the frontal threshold is used to determine to which ear, at a given frequency, the lateralization has occurred. One of the problems of using the Weber test for this purpose is that lateralization at the mastoid process is not always consistent with lateralization from the midcranium. The patient may lateralize the sound on the Weber test to the right ear. However, when the bone-conduction oscillator is placed on the right ear, he may report hearing the sound in the left ear. In still other cases, the patient will report hearing the sound on the left side at a given frequency when the oscillator is placed on the right ear, and on the right side when the oscillator is placed on the left ear.

Practices also vary considerably in the method of calibration of the masking noise used. In the former audiology clinic mentioned, the level of the masking noise used routinely in bone-conduction testing is 75 db. re 0.0002 microbar. At the second center referred to, where masking in bone-conduction testing is used only when the patient refers the test tone to the nontested ear, the arbitrary level on the masking dial contained in the audiometer used is indicated on the audiogram next to the frequency tested. The degree of masking used in this center is determined by the level required to cause the patient to localize the test tone back to the ear being tested. Many examiners who use those audiometers manufactured by the Beltone and ADC companies, which were designed by Ralph Allison, and the newer clinical audiometers manufactured by the Maico Company use a masking dial calibrated in terms of "effective masking," which refers to the level of the masking noise which renders a pure tone of a given intensity just inaudible when both signals are presented to the same ear. The masking noise contained in most of the Allison Hearing Evaluation Units and in the Sonotone Model 23 Advanced Clinical Audiometer is calibrated re: 0 db. equals normal hearing. These are several of the modes of calibration used for the masking noises incorporated in some of the currently available commercial audiometers.

None of the directors of the audiology clinics interviewed were satisfied with the procedure used in masking the nontested ear by air and particularly by bone conduction, and all felt strongly that further investigation of the problem was needed.

A series of 35 subjects were selected from among the patients referred to the Audiology and Speech Center of Walter Reed Army Medical Center having a differential in the hearing acuity of the two ears of 30 db. or more. A specially constructed audiotesting assembly consisting of two masking enclosures through which either pure tones or one of several masking noises could be introduced was used during the tests.

In the present paper, the specially constructed audiotesting assembly used in the tests will be described and the results ob-



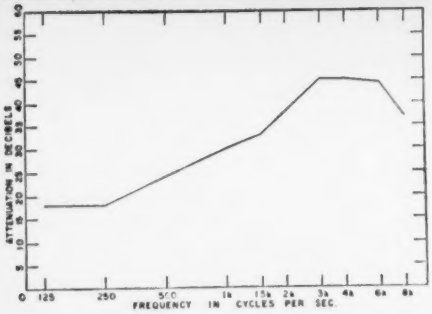
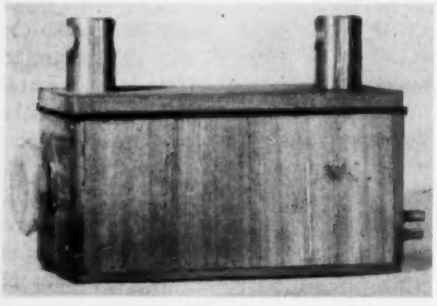


Fig. 1.—Above, semiplastic earphone sockets. Left, typical noise attenuation of the new earphone socket.

tained discussed. That part of the investigation in which thresholds obtained while the noninvestigated ears of the subjects were exposed to three currently employed masking noises will be described in a forthcoming paper.

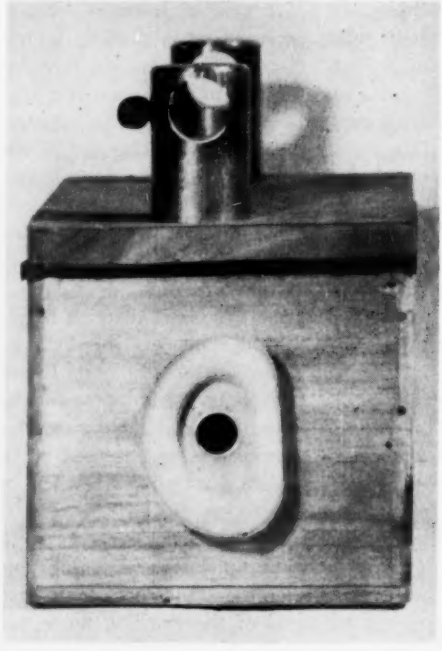
Fig. 2.—Exterior of masking enclosure, side view.



Description of Experimental Apparatus

Two large acoustically treated masking enclosures made of mahogany were constructed. The size of each enclosure was 13½ by 7¾ by 7½ in. At the outer end of each box was a loudspeaker which fed into a specially constructed model of a sound-treated room ending in a Zwislocki semiplastic ear socket. A picture of these ear sockets which were in direct contact with the subject's ears and the noise attenuation provided are shown

Fig. 3.—Exterior of masking enclosure, front view.



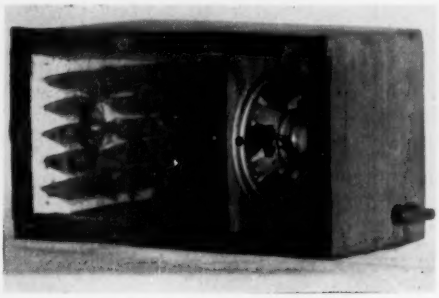


Fig. 4.—Interior of enclosure with lid removed.

in Figure 1, which is available through the courtesy of the Grason-Stadler Company.

The volume of the sound-treated compartment of each enclosure is 3,675 cc., which is considerably greater than the 2,000 cc. which Watson and Gales [11] found was sufficient volume to prevent spurious elevated bone-conduction threshold shifts at the test frequencies. This "pseudo sound isolated chamber" was completely lined with pieces of rug. Fiber glass was placed between a special type of sound-absorbent material known as "foam polyethylene." Acoustic data on this material are still restricted. It has a high average coefficient of absorption, and the noise-reduction coefficient is also high.

Figures 2 and 3 show two exterior views of one of the masking enclosures with the ear sockets in place. Figure 4 shows the enclosure with the lid removed, showing the loudspeaker and the specially constructed miniature sound-treated room. A simple supporting structure for the enclosures was constructed to make the apparatus rigid and stable. The boxes were adjustable both vertically and horizontally through a specially constructed spring arrangement so that they were in close contact with the patients' ears as shown in Figure 5.

Fig. 5.—Air-conduction testing with experimental apparatus.

The results of research conducted with similar equipment at other institutions will be briefly reviewed.

The use of this type of audiotesting assembly in pure-tone air and bone-conduction testing is not new. Norman Watson [11] constructed and used such an assembly in the Acoustical Laboratory at the University of California at Los Angeles. Watson employed this assembly in an attempt to overcome the difficulties and errors encountered in testing the hearing of a subject by means of the usual equipment and methods. He found that the use of these source boxes (1) eliminates room noises with levels up to 40-45 db. for a normal hearing person; (2) shields against the stray air radiation from the bone vibrator so that accurate bone-conduction tests can be made at high frequencies previously out of range for bone-conduction tests; (3) does not give rise to false, enhanced bone-conduction thresholds even at low frequencies, and (4) allows accurate air- and bone-conduction threshold measurements on either ear and is especially effective for testing cases in which one ear is normal and the other severely impaired. For his tests to determine the optimal masking level (thermal noise was used) for a tone of 250 cps, Watson used "a normal hearing person." These enclosures were also used on a subject having a severe mixed unilateral loss of hearing and on "a few hard of hearing persons, especially those with marked unilateral losses."

The use of a similar device was incorporated into the Petersen audiometer and is described by H. Jørgensen.[12] The M. P. Petersen audiometer is equipped with two spherical loudspeakers provided with insulating air-filled rubber rings. With this audiometer, it was possible to apply the same sound producer for the bone-conduction determination as is used for the air-conduction determination.

The possible value of routinely utilizing this equipment in clinical testing was considered in the present research. In view of the promising results obtained in the limited investigation which had been carried out on the several unilaterally deaf subjects reported in the literature, a more intensive study of a larger, carefully selected group of patients was considered necessary to make it possible to evaluate the value of this equipment for general, routine use in clinical audiometry.

Test Procedures

1. *Conventional Audiometric Procedures.*

The results of conventional audiometric procedures performed on all patients referred to the Audiology and Speech Center for audiologic diagnosis and rehabilitation are of importance in the present study because the results of these tests will be compared with results obtained when the experimental testing apparatus was used. No one test was considered conclusive, and all patients were seen for three visits separated by at least 24 hours. A different examiner saw the patient each time, so three audiologists had seen each patient.

The noise level of all testing rooms in the Acoustics Section was below 30 db. re: 0.0002 microbar (flat scale on General Radio Sound Level Meter No. 759B). Two Maico H1 Audiometers and a Maico E2 Audiometer were used for routine pure-tone testing. This testing included air-conduction measurements at octave points in the 125 to 8,000 cycle range, and bone conduction measurements at the five octave points in the 250 to 8,000 cycle range. When a difference of 30 db. or more existed between the two ears, air-conduction measurements of the patient's poorer ear were performed both with and without masking. The scores averaged were always the masked scores. Bone-conduction testing is performed with masking when there is evidence of cross lateralization of tones. This masking is of the sawtooth variety available with Maico audiometers and is chiefly low frequency in character. This noise is calibrated re: 0.0002 microbar.

Seventy-five decibles SPL (or more when needed) were used for masking in air-con-

duction testing and 60 db. or more for bone-conduction measurements when there was evidence of lateralization of tones to the nontested ear. When masking was used in bone-conduction measurements, both the masked and the unmasked thresholds were available for the "conventional" audiometric results. Neither the director of the Center, the chief supervisor, nor the supervisor of the acoustics section is completely satisfied with the masking procedure utilized, and it is for this reason that the present study was approved for conduct at the Center. Speech audiometry was performed with speech-reception thresholds and discrimination scores established for each ear individually and for both ears through earphones and in a free field. A special Stenger test, the story test, and, when indicated, the psychogalvanic skin-resistance test were used to rule out the presence of a nonorganic involvement. It is the results of these tests which were utilized in determining the diagnosis of the hearing loss present in the poorer ear by "conventional" procedures. The otologist's diagnosis was also available on each of the subjects included in the study. This information was compared with the results obtained with the experimental testing apparatus. When a patient was referred from another section of the hospital, he was retested by the investigator according to procedures conventionally used in the Acoustics Section of the Center.

2. *Experimental Testing Procedures.*— The testing period for each patient using the specially constructed audiotesting assembly was approximately three hours, divided into two one-and-a-half-hour periods. During the first testing period, satisfactory rapport was established with the patient, and the nature and significance of the research problem was explained to him. The following testing procedures were then carried out:

(*a*) Recheck air-conduction and bone-conduction thresholds on the poorer ear. The masking procedure used in the Acoustics Section of the Center was reproduced during this part of the test to maintain uniformity of procedure in checking results. The purpose of this portion of the test was to determine any possible changes in the patient's hearing acuity which might have occurred between the time of the initial testing and the research testing.

(*b*) Determine the Experimental Masking Levels (EML) for the seven octave points between 125 and 8,000 cycles for air conduction using the different types of masking noise introduced through an enclosure over the contralateral ear. These levels were established in one of three ways: (1) the level of the masking noise required to cause the subject to localize the test tone to the ear under test was used in those instances where the pure tone was lateralized to the nontested ear until a certain level of the masking noise was reached; (2) the level of the masking noise required to eliminate a shadow response was used for those subjects who continued to lateralize the test tone to the better ear until so much masking was introduced that the test tone was rendered inaudible, and (3) the level of the masking noise required to produce linear threshold shifts upward (greater loss) in the tested ear was used in those cases where the subject localized the tones to the ear under test.

(*c*) The masking enclosures were placed over the patient's ears with an attempt made to insure as tight a seal as possible without making the patient uncomfortable. This was easier to accomplish in the case of some patients than with others. The contours of the patient's head affected the ease with which a seal was secured. A leak between the lower portion of the patient's external ear and the semiplastic ear sockets was the most difficult to overcome. The patient's pure-tone air-conduction thresholds were then retested with the tones introduced through the enclosure on the poorer ear and the better, nontested ear covered with an enclosure through which no sound was introduced. Appropriate corrections were made at each frequency for transmission loss across the boxes.

(*d*) The same procedure as above was reproduced except that full-spectrum white noise was introduced through the enclosure over the nontested ear at the levels established in Step (*b*). The thresholds thus obtained were recorded.

(*e*) The same procedure as Step (*c*) was followed except that filtered bands of white noise were introduced through the enclosure over the patient's better, nontested ear. These filtered bands of noise were composed of frequency components one-third of an octave above and one-third of an octave below the test frequency. The level at which these bands were introduced was the EML established in Step (*b*).

(*f*) The same procedure as above was reproduced except that the noise available with the Allison Hearing Evaluation Unit was introduced

at the EML into the enclosure over the better ear of the patient. This was the last procedure performed in the air-conduction test group.

(*g*) The present step began the second hour of testing. The bone-conduction tests performed involved placement of the bone-conduction oscillator over the mastoid prominence of the temporal bone of the patient's poorer ear. This is not necessarily the most desirable place to place the oscillator. Indeed there is considerable evidence that there are more satisfactory locations for the placement of the oscillator.[7] It was not considered within the province of the present study to determine the most suitable location, and therefore the conventional position used in clinical audiometry was employed. The bone-conduction oscillator used was of the hearing aid variety. With the oscillator placed on the mastoid prominence, EML's for the different types of masking noises were determined with an enclosure covering the nontested ear. The thresholds thus obtained were recorded. As with air-conduction measurements, there was difficulty in determining the "true" bone-conduction thresholds of the poorer ear, an important limitation of the present study.

(*h*) The patient's bone-conduction thresholds for the poorer ear were determined with an enclosure over the nontested ear but no masking noise introduced into the noninvestigated ear. The nonenclosed bone-conduction thresholds were available from the results of "conventional" testing. Where masking was used in bone-conduction testing, both masked and unmasked thresholds were available.

(*i*) B. C. thresholds for the poorer ear were determined with full-spectrum white noise introduced into an enclosure over the nontested ear at levels established in Step (*g*).

(*j*) Filtered bands of white noise composed of a third of an octave above and below the test tone were introduced through the enclosure over the patient's better ear and the thresholds obtained recorded.

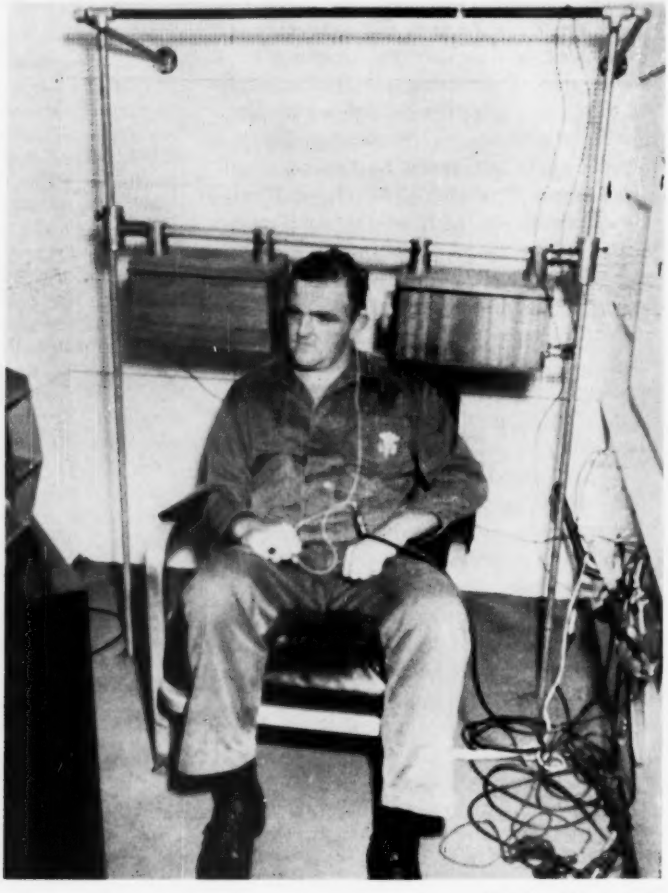


Fig. 6.—Bone-conduction testing with experimental apparatus.

(*k*) Allison complex wave masking noise was introduced through the enclosure over the patient's better ear and the thresholds thus obtained recorded.

Pictures of a patient during the first and second testing periods are shown in Figures 5 and 6. In Figure 5, the subject's air-conduction thresholds on the poorer ear are being established with a masking enclosure over each ear. Pure tones at the octave points between 125 and 8,000 cycles are being introduced through the enclosure over the patient's right ear. Different types of masking noises are simultaneously being presented through an enclosure over the patient's nontested ear. The examiner viewed the patient through the glass window at the patient's left. Figure 6 shows the same patient during the second period of testing. The bone-conduction oscillator is on the mastoid prominence of the patient's left ear. A masking enclosure covers his nontested ear, through which different types of masking noises are introduced.

Experimental Control

The experimental control utilized the results of conventional audiometric procedures and the otologist's diagnosis of the type of hearing loss present. The results obtained with use of the experimental apparatus on each of the subjects was compared with the results of "conventional" audiometric procedures to determine differences, if any, in the type of loss in the subject's poorer ears shown by the two testing approaches. When a significant difference was found between "experimental" and "conventional" testing, the otologist's diagnosis was checked to determine whether the new (experimental) results were more or less consistent with the medical diagnosis than were the results of the conventional audiometric procedures performed. The limitations of this control and of other aspects of the investigation will now be considered.

1. In the case of some of the subjects, the test tones were localized to the ear under test, and the point where threshold shifts occurred in the presence of the masking noise was used as the basis for determining the EML. The response given by such subjects without masking may represent what they are actually hearing in the ear being tested and masking in these cases may be unnecessary. The failure of the tester to obtain threshold shifts in the investigated ear until a certain masking level is reached does not mean that the tones are being heard only in the nontested ear. Both ears may be contributing to the subject's responses throughout the test.

2. The results of using the specially constructed experimental assembly on a group of subjects are compared with the testing procedures performed at the Audiology and Speech Center and are designated as "conventional procedures." Conventional procedures as performed at this institution are not necessarily typical of audiometry performed elsewhere. I feel that the audiometry performed at Walter Reed is at least as good as that performed at other institutions. We cannot say, however, that other examiners using different testing procedures at other institutions would arrive at the same results as were found by the clinical audiologists at Walter Reed. It is entirely possible that where a discrepancy was found between the types of loss indicated by "conventional" and "experimental" testing, an examiner at another institution might have found the same result as was obtained by the experimental testing. The comparison, therefore, between conventional and experimental procedures is limited by this factor. Conventional testing in this study refers only to the audiometric procedures used at Walter Reed during the time that the study was in progress. The superiority of the experimental assembly over conventional audiometry, therefore, is not generalized by the present study, in which are described the results of testing with a type of apparatus not in general clinical use at the present time compared with routine procedures used at a leading audiology center.

3. The basis for determining the consistency of a diagnosis arrived at by using the experimental apparatus was to compare these findings and those arrived at by "con-

ventional" procedures with the otologists' diagnoses. Otological reports on the etiology and type of deafness are often vague and incomplete and sometimes arrived at by a process of elimination rather than by any positive findings (e. g., otosclerosis). Such a comparison is therefore limited by the manner in which many otological diagnoses are established and stated. After consideration of a number of possible controls, it was felt that the use of these diagnoses was the best that was available in arriving at some sort of rough evaluation of the results obtained. Therefore, while we cannot conclude on the basis of the present limited study that the experimental testing assembly used is superior to presently available clinical procedures, the results are suggestive of what appears to be a promising approach to pure-tone audiometry in cases of the type tested in the present study.

Results of Tests Performed

Of the 35 subjects in the study, 31 were administered the complete battery of tests with the experimental apparatus described. Two of the three of the original group of thirty-five patients were disqualified because further tests for nonorganic deafness revealed a significant nonorganic component, and the residual organic loss of hearing was not of sufficient severity to require the use of any type of masking in the conduct of the audiometric test procedures.

1. *Instances Where the Use of the Experimental Apparatus Confirmed the Results of Conventional Audiometry.*—In 17 of the 31 subjects tested, the results of using the experimental apparatus confirmed the results of conventional audiometry for both air- and bone-conduction measurements. In all of these cases there was good consistency between the results of conventional audiometry and the examining otologist's diagnosis. The results of using the conventional audiometric procedures described will henceforth be designated as CAP (Conventional Audiometry Procedures); those obtained when using the specially constructed audiotesting

TABLE 1.—*Instances of Agreement Between CAP and EAP*

Type of Loss	Number of Cases	Per Cent of Total Number
Conductive	6	19
Sensorineural	7	23
Combined or mixed	4	13
Total	17	55

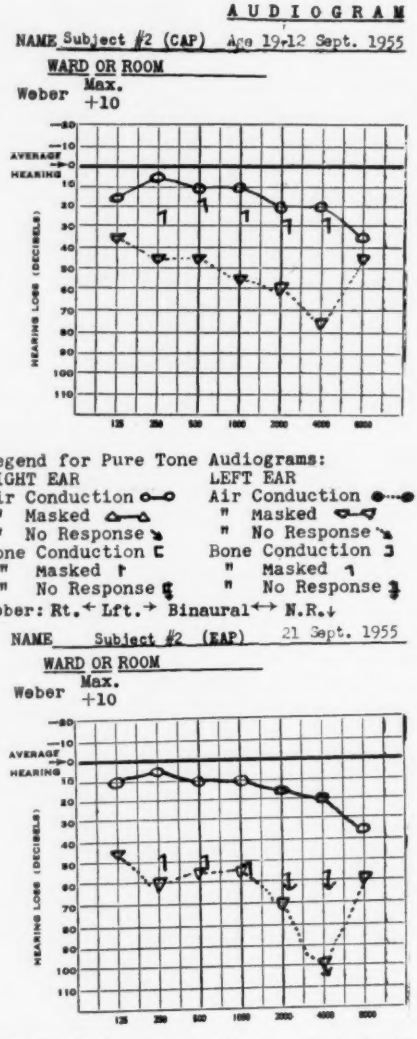


Fig. 7 (Subject 2).—Audiograms obtained with conventional audiometric procedures (CAP) and experimental audiometric procedures (EAP).

assembly by EAP (Experimental Apparatus Procedures). The types of losses in which EAP and CAP were in agreement were distributed as shown in Table 1.

2. *Representative Instances Where the Use of the Experimental Apparatus Produced Different Results from Those Obtained When Conventional Audiometry Was Used.*—(*a*) CAP: combined; EAP: sensorineural. Subject 2 whose audiograms are shown in Figure 7 was a 19-year-old army private who reported a loss of hearing mostly in his left ear since firing the M-1 in basic training five months before the date of examination. Otological examination showed essentially negative findings with the loss of hearing due to acoustic trauma incurred during basic training. Conventional audiometry showed a moderately severe combined loss in the left ear averaging 53 db. for air conduction and 25 db. for bone conduction. There was nothing to account for a conductive component in the patient's loss of hearing. EAP showed a predominantly sensorineural loss of moderately severe degree averaging 63 db. for air conduction with no significant conductive component, bone-conduction readings closely approximating the air-conduction curve.

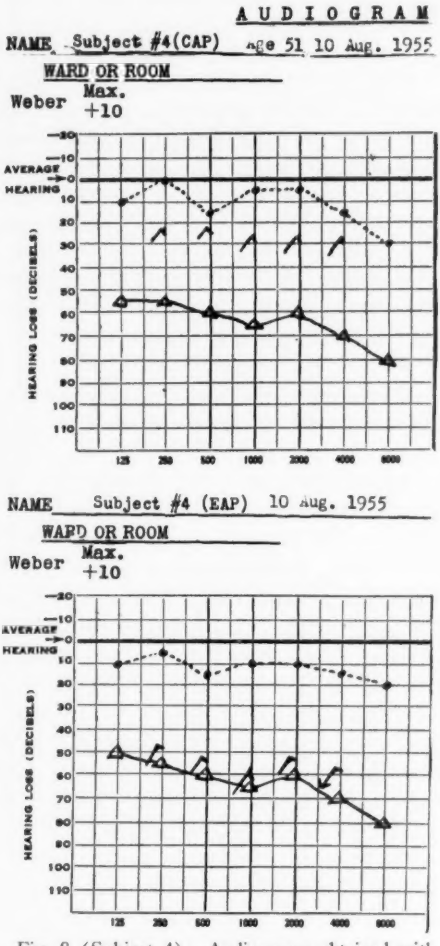


Fig. 8 (Subject 4).—Audiograms obtained with conventional audiometric procedures (CAP) and experimental audiometric procedures (EAP). (For explanation of symbols see Figure 7.)

Subject 4, whose audiograms are shown in Figure 8, was a 51-year-old woman who complained of a loss of hearing and tinnitus in the right ear for about 15 years. She had had intermittent dizzy spells for 15 years and was receiving a complete diagnostic work-up in the main section hospital. ENT examination was essentially negative, with nothing in the case history to account for a conductive loss. EAP showed a predominantly sensorineural loss with an overage air-conduction loss of 63 db. The examining otologist felt that Ménière's disease should be considered, but he did not consider this a true case of the disease. He believed the dizzy spells were probably associated with the menopause which the patient had been going through for the last seven years.

(*b*) CAP: conductive; EAP: sensorineural. Subject 6 was a 20-year-old private, first class, who reported a loss of hearing in his right ear starting with scarlet fever at the age of 5 years. There was no known otitis media associated with the disease. Otological examination was essentially negative. CAP showed a predominantly conductive loss of hearing in this ear and EAP showed a predominantly sensorineural loss. When tones were presented to the right ear with the bone-conduction oscillator over the mastoid prominence and a masking enclosure placed over the nontested ear, the

patient reported difficulty in telling in which ear the tones were being heard, with the 1,000 cycle and 2,000 cycle tones apparently heard in the left or nontested ear. Further testing with the experimental apparatus using experimental masking levels of full-spectrum white noise, filtered bands of white noise, and Allison noise showed a marked bone-conduction loss at all frequencies tested. These results were clearly at variance with those of the conventional audiometry performed. Scarlet fever at the age of 5 years seemed to be the only etiological factor of significance, with possible acoustic trauma sustained during basic training.

Subject 20 was a 26-year-old private, first class. The patient reports having difficulty in hearing with his left ear as long as he can remember. He was born two months prematurely. No tinnitus or pain in the ear was present, and there was no history of middle-ear infection. The otologist's diagnosis was essentially negative, and a diagnosis of congenital nerve deafness was made in spite of audiometric results suggestive of a conductive lesion. The mere presence of the masking enclosure over the nontested ear during the bone-conduction test was sufficient to cause the patient to localize the tones to the ear covered by the enclosure. When the different masking noises were introduced through this enclosure at the critical levels previously established, the loss in the poorer, examined ear appeared to be clearly perceptive in nature. This EAP finding was considered to be in good agreement with the otologist's findings and diagnosis.

Patterns of Tone Localization

The chief effect of the masking enclosures seems to be in exposing a previously established bone line as spurious. Losses previously considered conductive or combined when conventional procedures were utilized were, in some of the cases tested, shown to be sensorineural. The enclosures seem to encourage a lateralization of bone-conduction sounds to the nontested ear in those cases where the mechanism of shadow hearing is in operation, i. e., when the bone line on a given ear is a reflection of the hearing in the ear with the better cochlear reserve. In these cases, localization with earphones had been to the tested ear.

To what extent can the patient's reports of lateralization of a bone-conduction tone to the nontested ear be relied upon? These patterns of lateralization seem in some cases to be very bizarre and inexplicable. A number of audiograms were examined in the course of the present study in which the bone conduction tones were lateralized to the poorer ear when the better ear was being tested. Apart from the fact that the entire skull is stimulated whenever a bone-conduction oscillator is placed on the mastoid prominence or anywhere on the skull, the phenomenon of referred sensation as it applies to the mechanism of shadow hearing needs to be investigated. A number of such patterns of localization were found by the examiner, and a differential between the two ears by bone conduction of 10-15 db. need not be present for this phenomenon to occur.

Several of the patients tested reported finding it easier to localize tones when the enclosures were placed over the ears, compared with testing with earphones. In a number of the cases tested, the presence of the masking enclosure over the nontested ear during bone-conduction testing was sufficient to cause the subject to localize the tones to the nontested ear without any masking sound introduced through the enclosure. In previous conventional testing with earphones, the sounds had been localized to the tested ear. In these cases, it was possible to expose a previously established bone line as a shadow curve by the lateralization of tones without masking to the nontested ear. The loss using the experimental apparatus was either combined or sensorineural in these cases rather than conductive. In such cases the EAP results were in good agreement with otological findings and diagnoses.

TABLE 2.—*A Comparison of Experimental and Conventional Testing Procedures*

Type of Procedure	Agreed	Disagreed	Agreed and Disagreed
Conventional Test	17	14	31
Experimental Test	31	0	31
Combined	48	14	62

Summary of Findings and Statistical Interpretation of Results

The results of testing the 31 subjects in the study with the experimental apparatus are summarized in Table 2. "Agreement" means that testing with the experimental apparatus coincided with the otologist's diagnosis; "disagreed" means these results did not coincide with the medical report.

These findings are of statistical significance and indicate that only 1% of the time could the differences which were found between experimental and conventional procedures have occurred by chance. The differences obtained between the results of using the conventional and experimental procedures must therefore represent differences in some characteristic of the experimental procedure.

Summary and Conclusions

A simple audiotesting assembly was constructed consisting of two wooden enclosures which were used to present different types of masking noises to the nontested ears of each of the subjects in the study. The thresholds obtained on the poorer ears of the subjects when the masking enclosures were used were compared with those obtained when conventional audiometric procedures using earphones were employed. It was found that the results obtained when the specially constructed audiotesting assembly was used were in generally better agreement with the otologists' findings and diagnoses than were those of conventional audiometric procedures when the two testing approaches showed different results.

The audiotesting assembly employed in the present study appears to affect the pattern of tone localization in bone-conduction testing of some of the subjects having the requisite differential of 30 db. in the hearing acuity between the two ears. By encouraging a lateralization of the test tones to the ear having the better bone conduction covered by the enclosure, a spurious bone-conduction curve which represents a reflection of the subject's hearing in the better ear may be uncovered. The mechanism by which this occurs is not clear, but the presence of the enclosures over the subject's ears appears to encourage a lateralization of test tones, particularly in bone-conduction testing, to the noninvestigated ear when the mechanism of shadow hearing is in operation. This phenomenon occurred in these cases only when enclosures were used and did not occur in conventional testing with earphones. Should other investigators find a similar result with this type of testing apparatus, we may have a useful clinical instrument to aid the otologist in arriving at a more accurate differential diagnosis between conductive and sensorineural losses in those cases where a considerable discrepancy exists between the hearing sensitivity of the two ears.

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Case Reports

Extracranial Aneurysm of the Internal Carotid Artery

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Aneurysms of the extracranial portions of the internal carotid artery are rare, and, of the 85 cases reported, in all but 2 the patients were less than 15 years old. We are adding to the reported cases that of a woman aged 50 years who made a complete recovery after surgical extirpation of the aneurysm.

The patient, a Negro woman 50 years of age, had been seen in the Medical Clinic since 1949 because of epileptic seizures. She had been having these seizures since the age of 23 years, at which time she was involved in an accident and became unconscious. She had been taking medication regularly and her last seizure was Dec. 10, 1955. An electroencephalogram revealed an abnormal tracing suggesting epilepsy.

On her visit to the clinic on Dec. 10 the patient complained of a lump in her throat, which she stated had been present for seven years. Within the previous two months it had become larger and caused her to have slight difficulty in breathing through the left side of her nose. The patient was therefore referred to the ENT clinic for evaluation.

On examination the ears, nose, and throat were essentially negative, except for a smooth regular mass in the left oropharynx just posterior to the palatopharyngeus muscle and extending just above the level of the soft palate. During physical examination the patient was apprehensive and uncooperative; however, a firm sharply demarcated mass could be palpated (Fig. 1). There appeared to be a very little pulsation. Six consulting members of our clinic felt that this was a tumor which should be biopsied. The patient was admitted to the hospital on Dec. 26 for study. At that time the complete blood count, coagulation time, clot reaction time, prothrombin time, fasting blood sugar, blood urea nitrogen, blood chlorides, blood sodium and potassium, A-G ratio, and thymol-turbidity were normal. Two separate blood Wassermann tests were nonreactive. Urinalysis was normal. ECG suggested an old healed inferior myocardial infarct. The x-rays of the pharynx revealed a well-defined soft tissue mass of the region of the left posterior oropharynx. A skull x-ray was negative. A chest x-ray showed slight prominence of the trunk markings throughout both lung fields, suggestive of tracheobronchitis. The heart was slightly enlarged in the region of the left ventricle, and there was moderate elongation and tortuosity of the aorta, associated with slight calcification in the arch of the aorta.

Fig. 1.—Extracranial aneurysm of the internal carotid artery presenting on the left side of the pharynx.

On Dec. 28 the patient was taken to the operating room and under local anesthesia an attempt was made to biopsy the mass. The patient was uncooperative, and several small superficial pieces of tissue were removed with a punch. This was

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From the Department of Otolaryngology, Graduate Hospital of the University of Pennsylvania.

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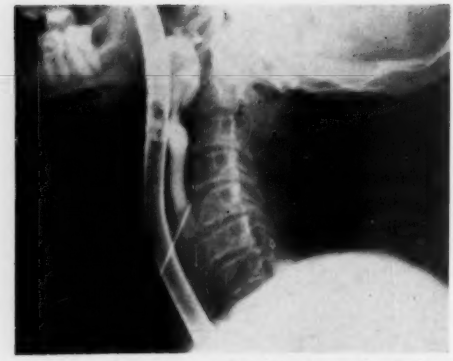

Fig. 2.—Arteriogram showing size and relations of the aneurysm.

reported by the pathologist as fragments of tissue representing chronic pharyngitis.

On Jan. 4, 1956, the patient was again presented to the ENT conference. The majority opinion was that the biopsy was too superficial and that this was most likely a mixed tumor of the salivary glands. It was recommended that the patient be given a general anesthetic and that the tumor mass be removed as completely as possible as a biopsy specimen.

On Jan. 6 while the patient was under general anesthesia and completely relaxed, the mass was palpated and a marked pulsation was felt. A No. 22 needle was inserted into the mass, and a free flow of blood was obtained. We felt rather certain that this was an aneurysm or a vascular tumor, and therefore the surgical procedure was abandoned at this time. An arteriogram was taken on Jan. 12 (Fig. 2), which revealed a large aneurysm about 3.5 cm. in its greater diameter arising from the internal carotid artery just distal to the bifurcation.

The patient was then prepared, and adequate blood reserves were secured for removal of the aneurysm and possible arteriograft to the left internal carotid artery. On Jan. 16 under general anesthesia an incision was made in the left side of the neck at the medial aspect of the sternocleidomastoid muscle (Fig. 3) parallel with it from the mastoid tip to the lower border of the thyroid cartilage. The superior belly of the omohyoid was divided and the internal jugular vein exposed. The vein was ligated and removed in order to fully expose the common carotid artery. The common carotid artery was exposed at the level of the thyroid cartilage. The vagus nerve was identified and a tape passed around the common carotid artery in order to occlude the vessel in the event of uncontrollable bleeding. Upward dissection of the common carotid artery was then continued. In order to expose the bifurcation completely, the intermediate tendon of the digastric muscle was divided, together with the stylohyoid muscle, and a skin incision was then made beneath the mandible toward the midline. The mandibular branch of the facial nerve was identified and reflected with the skin flap. The carotid bulb was exposed, and the external and internal carotid trunks were identified together with the carotid bulb. The hypoglossal nerve and the descending hypoglossal nerve were retracted forward. The branches of the external carotid were identified, and the superior thyroid, ascending pharyngeal, lingual, and facial arteries were ligated so that the external carotid could be retracted to provide proper exposure of the internal carotid artery. The internal carotid was identified, and a heavy black silk suture was placed around it just above the carotid bulb. As the dissection was carried upward approximately 1.5 cm. distal to its origin, the aneurysm, which measured approximately 4 cm. wide by 6 cm. long, was encountered deep within the retropharyngeal fascia. By dissection the aneurysm was isolated. The glossopharyngeal nerve was encountered and retracted forward from the field (Fig. 3). The upper limit of the aneurysm was determined and a heavy black silk tie placed around the artery. On compression of the proximal end the pulsation almost completely ceased. The internal carotid was then clamped proximal and distal to the aneurysm and the aneurysm was excised. The ends of the artery were suture-ligated and tied. The aneurysm contained a large organized thrombus. Hemostasis was then maintained, and the wound was closed with a Penrose drain in place. The patient developed marked edema of the oro- and hypopharynx; therefore, a tracheotomy was performed.

The day after the operation, Jan. 17, 1956, the patient was examined and found to have a hypoglossal paralysis of the left side, with a posticus paralysis of the left recurrent laryngeal nerve.

Fig. 3.—Internal carotid artery showing lower portion of the aneurysm.

The patient progressed satisfactorily postoperatively. On Jan. 27 the tracheotomy tube was plugged, and the following day it was removed. At this time she had regained almost complete recovery of the left recurrent and hypoglossal paralysis and, although the airway was adequate, the patient had slight hoarseness, which later abated.

At no time was there any evidence of hemiparesis; however, we felt there was a change in the patient's personality. Preoperatively, she had a quiet, withdrawn personality, whereas postoperatively her personality was that of an extrovert. The patient was discharged on Feb. 3, 1956, with no complaints. She had not taken any medication for her epileptiform seizures when last seen and has not had any attacks.

Pathology report*: "The specimen consisted of several fragments of tissue which were irregular in shape and had a marked variation in size. The largest one was in the shape of a sac grossly resembling an aneurysm sac, measuring 3 by 2.5 cm. All of the fragments showed a similar appearance, being gray or gray-white in color. Many of them had areas of calcification intermingling with areas of a soft consistency. The surface of one side of each fragment was smooth, and yellow in color, while the opposite side represented the internal wall of the vessel and was irregular and calcified.

Microscopically several sections were examined, and they showed a wall of an artery modified by two types of lesion. One of them especially involved the intima, which appeared to be thickened and to be composed of a dense hyaline connective tissue which is characteristic of arteriosclerosis. In addition the media shows several deposits of a purplish-blue material resembling calcium, and the adjacent adventitia tissue is increased in thickness and shows mild perivascular infiltrations around the vasa vasorum. In the same specimen, sections of a lymph node show mild hyperplasia of the lymphoid tissue, with moderate congestion in the sinuses.

"Pathologic diagnosis: Arteriosclerotic aneurysm of the carotid artery."

One month after this patient's discharge, we saw a 60-year-old Negro woman in our clinic with a similar pulsating mass in identically the same position as that of the case just reported. Her only complaint was a lump in her throat, but the patient refused admission for study and treatment.

In reviewing the literature, we find that aneurysms of the extracranial portions of the internal carotid artery are rare.[1] Aneurysm of the common and the external carotid arteries have been reported more frequently, and trauma is stated to be a common cause, owing to their more exposed location. Aneurysms of the external carotid artery have been reported chiefly in war surgery, whereas aneurysms of the extracranial portion of the internal carotid artery have more frequently been reported in connection with infections of the oropharynx, particularly the tonsil and peritonsillar abscesses, and infections of the nasopharynx. Of 85 cases of aneurysm of the extracranial internal carotid artery reported, all except 2 have been in children below the age of 15 years. One of these occurred in a man 20 years of age and the other in a woman 70 years of age, both of whom had infection of the oropharynx. There has been no mention of this type of aneurysm due to arteriosclerosis, but we are certain there must be others similar to this case.

Probably the earliest reports suggesting the presence of aneurysm of the extracranial internal carotid artery are found in the reports of Kennedy,[2] 1834-1842. During the Dublin scarlatina epidemic, he reports three deaths from erosion of the vessels in the neck. Having observed many thousands of cases with only three hemorrhages, he remarked, "Of all parts of the neck, none appeared to me to resist the sloughing process so well as the arteries and salivary glands." This seems to be quite rare, and since only the fatal cases are reported, the comparative frequency of aneurysms of the extracranial internal carotid must be considered. On many occasions a small mass or lump in the throat may be neglected because of the mild nature of the symptoms.

Salinger and Pearlman,[3] in discussing hemorrhages from the abscesses of the throat, state: "In 90 postmortem examinations the vessel found to be most frequently involved was the internal carotid artery—49 times." This being the case, the number of aneurysms which have occurred and have thrombosed, with spontaneous recovery or have not come to autopsy, must be fairly common and difficult to estimate. As

*Courtesy of Pathology Department, Graduate Hospital, University of Pennsylvania.

Young[4] states, "The future proportion of all types of aneurysms is difficult to estimate, as the majority of the non-erosives do not depend on purely diseased phenomena. Traumatic aneurysms are mostly connected with warfare. The syphilitic ones must be diminishing in number, though they also may become more common after the deteriorated social conditions of war time." Of the cases reported other than from warfare, the majority have been in children, so we must not minimize infections of the pharynx as a prevalent cause.

The symptoms and signs depend on the etiology of the aneurysm. In most cases the patient complains of a lump in the throat. This may be the only complaint, as was noted in the two cases we have seen. Looking in the pharynx, one sees a tumor mass pushing the tonsil or posterior pillar inward and toward the midline or even across the middle of the pharyngeal cavity. According to Young,[4] Winslow states, "Aneurysms of the internal carotid artery rarely point in the neck." Approximately 40 cases of bleeding from the ear have been reported, all of them in children. Detailed descriptions of this mechanism have been given by Young and Harrison, where the aneurysm breaks into the lateral pharyngeal space through the parotid fascia, into the parotid space, and through the fissure of Santorini, into the external auditory canal. If the aneurysm is due to a septic process, there may be marked anemia, pyrexia, toxemia, and secondary manifestations such as torticollis, dysphagia, and dyspnea. There may be swelling behind the angle of the jaw, and pain may be vague or throbbing, increasing as the mass enlarges, owing to tension on the fascia. There may be pain in the ear, especially if the aneurysm breaks into the external auditory canal. In the early stages or just after a leakage, the mass is nonfluctuant, brawny, somewhat tender, but not discolored. It is usually pulsating, and a bruit is seldom heard because the aneurysm is deep behind the hematoma.

If the aneurysm is secondary to an abscess of the peritonsillar, parapharyngeal, or retropharyngeal region, the diagnosis may be difficult. Unless one is aware of this fact he may innocently incise the aneurysm, mistaking it for an abscess. Bleeding from the pharynx or nasopharynx invariably will occur sooner or later. The origin is sometimes very difficult to determine. Paresis of the palate, of the tongue, and of the cervical sympathetics, due to the inflammatory process, usually follows.

In treatment surgical intervention is advised, that is, surgical ligation of either the internal carotid, proximal and distal to the aneurysm, or ligation of the common carotid and the distal portion of the internal carotid. If extirpation is not possible, the next best thing is packing of the neck, which causes partial occlusion of the arterial stream, allowing periarterial fibrosis. Surgical delay increases morbidity and mortality.

Since ligation of the internal carotid, especially in younger people, carries with it a great morbidity and possibility of hemiplegia, an arterial grafting whenever feasible should be undertaken. The pharyngeal wall approach is not recommended, owing to its inaccessibility.

The differential diagnosis should include mixed tumors of the pharynx, lymphomas, leiomyomas, peritonsillar and retropharyngeal abscesses, carcinomas, and carotid body tumors. Aneurysms should be considered before surgical intervention is undertaken.

Summary and Conclusion

A case of arteriosclerotic aneurysm of the extracranial portions of the internal carotid artery has been presented. In this case the patient allowed treatment by surgical extirpation with complete recovery. In a second case suspected the patient refused treatment.

We have correlated our findings with those found in the literature of extracranial internal carotid aneurysms, most of which have been of septic origin, so that in considering a diagnosis, one may be more cautious in performing a biopsy of a pha-

ryngeal mass or incising a pharyngeal abscess.

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Progress in Otolaryngology

Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Internal Ear

Summaries of the Bibliographic Material Available for 1954

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A very extensive literature on the subject of chronic progressive deafness and its ramifications was surveyed for the year 1954. Professor Michel Portmann surveyed the French Literature and Dr. Bellucci the Italian. Portions of abstracts appearing in *Excerpta Medica* were utilized when necessary to cover the world literature adequately. The review has become very lengthy, even though we used less than half of the papers abstracted. Important new clinical information and significant research contributions form the bulk of the material.

Otosclerosis

Walsh[287] examines the evidence relative to the effect of pregnancy on deafness caused by otosclerosis. The assumption of a relationship between pregnancy and deafness, caused by otosclerosis, is mainly based on the history given by the patient. A review of 601 case histories collected by the author and two other observers shows that more than half of all women (52%-57%) reported no effect of pregnancy on hearing. More reliable information can be obtained by audiometric examination of patients who became pregnant following fenestration operation. In 72 cases observed by Day and Sullivan, there was only one in which progress of otosclerosis occurred with pregnancy. In the author's own series of 40 patients, only 2 showed a loss of hearing in the ear that had been operated on with subsequent pregnancies; one of these must be attributed to nerve damage; in the other, hearing returned after pregnancy to the prepregnancy level. Seven cases had a hearing loss in the ear not operated on, but closer analysis demonstrated that the deficit developed over a period of three to five years after pregnancy. Actual measurements of hearing in 110 women showed that there was progress of the disease in the ear that had been operated on in 12% of the cases. Physicians should realize that pregnancy is not responsible for deafness associated with otosclerosis. In this way much psychological trauma will be avoided. There is no excuse for advising therapeutic abortion in a woman with otosclerosis.

Fowler[94] furnishes additional data in support of the thesis that emotional factors are involved in causation, exacerbation, and extension of otosclerosis. Among siblings in families with a history of otosclerosis the incidence of the disease was found to be higher in the emotionally sensitive or unstable than in the more stable persons. In five pairs of identical twins, deafness due to otosclerosis developed first in the one who prior to onset of deafness had been the more emotional of the two. In two other

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pairs otosclerosis developed at about the same time in both, following severe emotional distress in their teens. Fowler cautions against a hasty diagnosis of deafness due to otosclerosis in children and adolescents. Often the hearing loss is due to some other lesion affecting middle-ear impedance. A false or unwarrantedly pessimistic diagnosis may cause emotional repercussions and prove more detrimental to a patient's health and hearing than the lesion.

Two pairs of identical twins with otosclerosis are reported by Hall and Ogilvie,[123] increasing the reported number of such pairs to a total of 21. The pieces of bone removed at fenestration from one pair of twins are structurally normal, but those obtained from the other pair show the quiescent stage of the diffuse form of otosclerosis and are remarkable for the essentially similar architecture. Emphasis is laid on a probable developmental origin as the etiology of otosclerosis.

The mechanism of tinnitus in otosclerosis is due to the perverted relationship between bone and air conduction, according to Saltzman.[249] Restoration of good air conduction by the fenestration operation produces a masking effect on the sensitive bone conduction of these patients. The fenestration operation may be offered with greater confidence to otosclerotics with some neural degeneration. The operation gives in the majority of these cases some improvement in hearing and considerable relief from tinnitus. Contraindications include advanced age, poor health complicating irritative labyrinthitis, and total or subtotal deafness of the other ear.

Sound transmission in clinical otosclerosis was studied by Rytzner.[246] Audiometric studies were made before, during successive stages of, and after fenestration surgery. This surgery has been of enormous benefit, but restoration of normal hearing has so far not been successful even in ears with normal cochlear function. This is due to modifications of the sound-transmission mechanism. A number of factors are important in order to obtain sufficient improvement in hearing from fenestration surgery. Blocking by various materials at the tympanic membrane can result in significant improvement. The larger the fenestra the better the hearing. A minor but significant improvement is obtained for speech frequencies following removal of the endosteum in the fenestra.

A distinctive pattern of response to work-hearing tests in uncomplicated otosclerosis is reported by Saltzman.[248] At moderate intensities the score was poorer for words of low-frequency range than for words of high-frequency emphasis. Word intelligibility at the average conversational level was poorer in otosclerosis than in nerve deafness and middle-ear disease. At 30 db. or more above the speech-hearing threshold, intelligibility approaches perfection in uncomplicated otosclerosis. Nor is there only an absence of recruitment, but there appears to be a slow growth loudness with moderate increments of intensity. The difference limen is large in otosclerosis.

Occlusion of the round window membrane without involvement of the oval window is discussed by Lindsay and Hemenway.[176] Such a case is presented with histological studies. Only 20% of the surface area of the round window membrane was free of the otosclerotic focus, yet hearing appeared to be good. If occlusion of the round window is capable of producing air and bone conduction thresholds similar to those found as a result of stapes fixation, some method for differentiating the two conditions is needed. The principle involved in the Gellé test offers a possible approach. The mobility of the stapes, as determined by testing with the probe during the fenestration operation, may not be a sufficiently accurate indicator of the efficiency of the mechanism as a sound conductor.

The serum cholinesterase level in relation to the neurovascular trouble of otosclerosis and the functional capacity of the autonomic nervous system is reported by Maggio.[72] The results secured have led the author to the following conclusions: (*a*) Cholinesterase activity is reduced by about 24%

in the serum of otosclerotics. (*b*) The reduction of serum cholinesterase in these cases may actually be in harmony with reduced general vagotonus and with a stronger action of epinephrine on the labyrinth which would in consequence produce paradoxical response of the organ to the action of the chemical stimulant in the nature of angiospastic phenomena in the neurosensorial organs and of capillary dilatation in the bony tissues which would be the immediate cause respectively of the appearance of the clinical aural syndrome and the osteoporosis in the bony capsule.

Falta [78] treated 15 female otosclerotic patients, ages 16-55, with a preparation consisting of 0.01 gm. testosterone acetate crystals, 0.1 ml. benzyl alcohol, in 1 ml. olive oil. In most of these patients other medications had remained almost completely ineffective. The hormone preparation was injected intramuscularly, twice weekly, for a series of 10-15 injections; after an intermission of two months the series was repeated. In 11 cases hearing was markedly improved and tinnitus considerably ameliorated. In two cases further progress of otosclerosis was controlled, while in two other cases no response whatsoever was obtained. Improvement was generally observed after the 4th or 5th injection; if there is no improvement after the 10th injection, at best, prevention of further decline of hearing can be expected. If necessary, a third series of 15 injections was administered six months after the second. Cases have been observed over a period of two and one-half years. The author reasons that testosterone increases the blood supply to the labyrinth, which in turn exerts a favorable influence on circulatory disturbances which are the cause of otosclerosis. The development of new foci of spongiosis is prevented, and in cases with marked improvement of hearing there were changes in existing foci; in particular immature bony substance and connective tissue were compensated. The series is too small to permit definite conclusions.

Palpation of the stapes through the external auditory canal can determine with certainty whether the stapes is partially or completely fixed, reports Rosen.[236] The partially fixed footplate of the stapes can be rendered more mobile and restore hearing. The completely fixed footplate of the stapes can be mobilized and restore hearing. Improved and even normal hearing may be obtained.

Mobilization of the stapes for otosclerotic deafness is presented by Rosen and Bergman.[237] Improved hearing was maintained 16-25 months, indicating that reankylosis of the stapes footplate had not occurred. A study of the cases in which the attempt to mobilize the footplate did not result in improved hearing suggests the following possible explanations: (1) an extreme degree of fixation which does not yield; (2) thin and fragile crura which fracture under the force required to mobilize the footplate; (3) faulty technique. If the direction of the force against the neck of the stapes is not in line with the crura, the latter may fracture. If the force is applied to the crus, which is thin, instead of the neck, which is thick, the crus may fracture. If for the above reasons hearing does not improve, fenestration can be performed without penalty. Two groups of otosclerotic patients appear to benefit by mobilization of the stapes: those with good cochlear reserve where socially usable hearing without a hearing aid can be obtained and those with mixed-type deafness where reversal of the conductive factor allows more efficiently aided hearing.

A critical survey of fenestration surgery is reported by Hutchinson.[143] The chances of obtaining a satisfactory result are greater in cases of chronic adhesive deafness than in cases of otosclerosis. The chance of success is much better in a pure case of either of these two conditions than when nerve degeneration is superimposed. In cases of otosclerosis deafness of less than five years' duration and less than 50 db. loss for the 2,048 frequency offers a good prognosis. In

adhesive otitis the duration should be less than 15 years and the loss for 2,048 be less than 60 db. At least two years should elapse after surgery before the result is evaluated.

Fenestration in the ideal candidate is reviewed by Farrior.[79] This is an otosclerotic patient with a flat or ascending air-conduction audiogram, with a 40-60 db. hearing loss for the speech frequencies, and normal bone conduction. Under average office conditions, a bone-conduction reading at the 15, 20, or 25 db. level is to be regarded as normal. In addition, another 5-10 db. drop at 1,000 and 2,000 (Carhart notch) is to be considered normal. Analysis of 100 mature 2-5 year postoperative cases showed 85% persistently good results above 30 db. Postoperative labyrinthitis is classified into primary surgical, secondary serous (inflammatory), and tertiary or degenerative cicatricial. The type, degree, and duration of postoperative labyrinthitis is measured with a calibrated Weber test.

According to Alexander,[3] the first pitfall of the fenestration operation is its performance in patients in whom it is not indicated. It is contraindicated when hearing loss is the result of nerve degeneration, or in the presence of gross pathologic changes in the middle ear. Adolescents and young adults are better candidates from the physical, emotional, and otologic point of view. The second pitfall is failure on the part of the physician to establish a close relationship with his patient. Absolute frankness is essential, particularly as to the nature of the disease, the operation, and its chances for success. The third pitfall is failure to undertake a complete physical and laboratory investigation before the operation. Another serious pitfall is omission to observe the most careful aseptic routine. Carelessness and complacency are a further pitfall. Every step must be performed with a technique as nearly perfect as it is humanly possible. In particular, the size of the skin flap should be accurately estimated so that it fits over the window like a glove. Bleeding is controlled before the fenestra is created. The next pitfall is failure to observe certain precautions to avoid accidents during the operation, the most frequent ones being linear tear of the plastic skin flap, and section of the chorda tympani. Not only visible fragments of bone, but also microscopic particles of bone dust should be removed in order to prevent osteogenic closure of the fenestra. Postoperatively careful aseptic technique must also be observed. The final pitfall is to set the standards too low. The minimum requirements for success are return to the 30 db. level in the conversational frequencies, and complete rehabilitation of the patient in his social and economic life.

Many problems in which the nonfenestrating otologist has the responsibility of advising his patient are answered by Farrior, Bagby, and Thomas.[80] The primary results of fenestration are largely dependent on the quality of the patient's bone conduction. Convalescence from fenestration surgery is usually pleasanter than that after any other major surgical operation. Significant permanent undesirable sequelae are extremely rare. Properly performed, the fenestration operation is relatively free from serious operative risks. The permanent patency of the fenestra and the permanent restoration of hearing are dependent solely upon the microscopic perfection of the surgical technique.

The chief obstacles to success in fenestration, reports Shambaugh,[261] were infection, osteogenic closure of the fenestra, and the sterile inflammatory reaction in the labyrinth. Research on animals helped considerably to solve the problem of closure and sterile labyrinthitis. Osteogenesis was found to be stimulated by bone dust, scrapping fenestral margin endosteum, and failure to enchondralize the area. Approximation of the skin flap to the endosteum further decreased osteogenic activity. Postoperative labyrinthitis was found to be severer in the presence of infection in the mastoid cavity; hence the need for meticulous aseptic technique. Sponge packing against the tympanomeatal flap decreased the postoperative labyrinthitis significantly.

A modified technique for fenestration is presented by the Antoli-Candela.[8] A step-like window with the superior edge higher than the inferior is made by removing the edge of the usual fenestra toward the facial nerve. The tympanomeatal flap now lies against this fenestra like a carpet on a staircase, insuring a close approximation with the endolymphatic labyrinth. The fenestra is covered with a free split-thickness graft of about the size of the classical tympanomeatal flap. It is thin, does not bleed, does not have adherent bone chips, and does not show postoperative inflammatory reactions.

Hearing results on 200 fenestration patients over a period of five years or more are presented by Campbell.[36] In a group of 130 successful cases, the average hearing for the three speech frequencies was only 0.5 db. worse at the end of five years than it was at the end of the first year. In 49 unsuccessful cases, an average loss of 6.4 db. occurred at the end of the fifth year. Factors affecting postoperative hearing are psychological reactions of the patient, inflammatory or allergic conditions of the nose and ears, secretory otitis, presence of tinnitus, emotional and nervous disorders, intralabyrinthine hypertension, auditory nerve degeneration, and further advancement of the otosclerotic process. Failures may be attributed to closure of the fenestra, postoperative serous labyrinthitis, wrong preoperative diagnosis, infections of the ear cavity, and persistent perforations of the drum. Cases with closure of the fenestra can be expected to have further losses in hearing due to continued advancement of the otosclerotic process around the stapes footplate.

Among 200 cases of fenestration reviewed by Vyslonzil,[285] the stapes appeared to be mobile in 16. These may have been cases of spasm of the tensor tympani muscle. In these cases, nevertheless, fenestration led to a good result. Damage of the membranous labyrinth did not bring about total deafness, and sometimes there was even lasting improvement of hearing. In damage of the membranous labyrinth, the disturbances of equilibrium were slight. This is explained by assuming that postoperative dropsy of the labyrinth which is very probably present is of slight influence, because of the overproduction of endolymph which may drain away via the fistula. There are wide variations in the transient postoperative reduction of the hearing capacity, every patient reacting differently to the operative trauma. These differences in reaction are probably related to the extension of the otosclerotic process. The fact that after administration of depressin, which causes a decrease of blood pressure, hearing is improved, even into the postoperative period of bad hearing, suggests that there is a postoperative hypertension in the labyrinth. After the blood pressure has become normal, auditory acuity is reduced again.

Shambaugh[260] discusses some of the special problems which he has encountered and overcome in fenestration procedures. Suppurative labyrinthitis can be avoided by use of a most careful and meticulously sterile technique reinforced by antibiotics. The incidence of osteogenic closure of the new window has been reduced to 1%-2% by attention to the following factors: 1. Every bone particle from the fenestra and surrounding area is meticulously removed by continuous flow of sterile Ringer's solution while the fenestra is being made, and by use of the operating microscope. 2. The endochondral bone, which regenerates more slowly than periosteal bone, is widely exposed. 3. As skin lying against bone tends to inhibit osteogenesis, the thin skin flap is snugly applied to the fenestral margins. 4. The periosteum and endosteum of the labyrinth are brought into approximation with the periosteum of the skin flap, assuring "healing by first intention." 5. The fenestra is finished by polishing with a smooth burnishing burr. 6. Local anaesthesia results in minimal bleeding and prevents infection. Postoperatively a hearing improvement of 5-10 db. can be obtained by loading the tympanic membrane with ointment or other material. Postoperative com-

plications are avoided by instituting leg and breathing exercises until the patient becomes ambulatory. Not all problems have been solved, but satisfactory restoration of hearing can now be achieved in 7 or 8 out of 10 cases.

Hexamethonium bromide procaine amide (to produce hypotension) and reverse Trendelenburg posture of the head are used by Fine, Bentel, and Meyerovitz [87] in controlled hypotension in fenestration operations. Systolic pressure is lowered to 50 to 65 mm. Hg. An excellent bloodless field is achieved by these methods. The dangers are (*a*) excessive fall of blood pressure with irreversible circulatory failure; (*b*) cerebral and coronary thrombosis; (*c*) renal failure; (*d*) liver necrosis; (*e*) blindness, and (*f*) reactionary haemorrhage. The dosage of hexamethonium varies from 50 mg. to 250 mg. plus 1 to 3 ml. of procaine amide. The hazards and the importance of skilled teamwork, both during the operation and in the postoperative management of these cases are stressed.

The location of the fenestra novovalis in the region of the ampulla of the horizontal canal has a definite influence on the improvement in the results of the fenestration, according to Miodonski.[197] The explanation of this by the fact that the fistula in the neighborhood of the ampulla is of somewhat larger dimensions and so closes with more difficulty, does not seem to be sufficient. From the general biological point of view a system of tissue tensions in the neighborhood of the ampulla comes into play, differing from that in the other section of the canal, i. e., different conditions govern the activity of the law of polar regeneration in these two sites.

Prevention of bone regeneration by cortisone and corticotropin (ACTH) in experimental fistulae of the labyrinth is reported by Mayer and Glaninger.[188] Guinea pigs were used in this trial of cortisone and corticotropin. Both substances were found to decrease bone formation, and of the two, cortisone did so better. Hearing improvement obtained through fenestration is mainly threatened by serous labyrinthitis and occlusion of the fenestra. According to Hussarek and Novotny,[142] these two complications are interrelated, as labyrinthitis heals with residual fibrosis which is followed by deposition of calcium and subsequently by bone formation. Others have shown that cortisone will prevent or inhibit postoperative serous labyrinthitis. Cortisone, furthermore, controls reactive connective tissue formation. In order to test the effect of cortisone, fenestration was performed in 15 adult guinea pigs, and cortisone was administered at different times postoperatively. If cortisone is started early and continued long enough, connective tissue formation and subsequent bony closure of the fenestra can be prevented. Applied to humans, it is suggested that cortisone therapy be started immediately with a daily dose of 100 mg., which will not produce vertigo. Medication should be continued 4-5 weeks. There are no side-effects, and only the well-known systemic contraindications against cortisone have to be watched. The time of follow-up was too short to ascertain whether bony closure takes place at a later date.

Many attempts have been made to find a method to prevent closure of the fenestra novovalis. A report is given by Gaini [100] of the results of an experimental study of acrylic resin for this purpose. The study was carried out on dogs. In order to prevent closure of the fenestra novovalis a stopper of acrylic resin with a canal running through it, was introduced. Histological examinations were carried out after a week, a month, and two months, and it was found that the acrylic resin, which is an inert substance, does not cause any particular reaction and that the use of a stopper of this substance prevents the formations of callus obliterating the fenestra novovalis, even though the canal is sometimes occluded by fibrous connective tissue. Moreover, when after some time the reparative processes are exhausted, one may remove the stopper, leaving a permanent opening.

Fabian [75] stresses the importance of the degree of pneumatization of the temporal

bone for selection of patients and prediction of results in fenestration operations. Three types of pneumatization are distinguished: A, patients with fully developed mastoid processes which are normally pneumatized; B, patients with insufficient or irregular pneumatization of the mastoid process; and C, patients with solid or spongy mastoid processes. In patients with ostosclerosis the incidence of Type A (good pneumatization) is much higher than in patients with inflammatory disease or in normal subjects. In turn, Type A shows a much more favorable response to fenestration operation. The factor of pneumatization plays no, or only a relatively little part when the prognosis for success of fenestration is good; in those with an altogether poor prognosis, however, it is one of the factors influencing the outcome of the operation. Poor pneumatization of the mastoid process is thought to be an additional constitutional defect. In patients with poorly pneumatized mastoid processes damage to the labyrinth during the operation is more likely, and there is a greater tendency toward hemorrhage in vessels close to the labyrinth. The conclusion is drawn that in patients with a doubtful or poor prognosis the finding of an insufficiently pneumatized mastoid process should suggest that hearing aid and lipreading are better suited for the social rehabilitation of the patient.

An extensive survey of the vestibular reactions of patients operated upon for otosclerosis is reported by Pursiainen.[225] Rotation tests were made by Bárány's method. In the postoperative tests the duration of postrotational nystagmus was shorter toward the operative side than toward the nonoperative. As regards duration of nystagmus, the difference between the two ears was greater in the postoperative tests, nystagmus being shorter to the side of the ear operated on. In none of the bilaterally fenestrated cases (7) was nystagmus elicited after the second operation, although the same patients had shown a positive response after unilateral fenestration when the test was made in the usual way with the head erect. The average duration and the latent period of caloric nystagmus were diminished after operation. The fistula test produced nystagmus in 135 out of 183 cases; the direction was homolateral in 112 cases, heterolateral in 18 cases, and varied between the two sides on repetition of the test in 4 cases; in 1 case there was a vertical nystagmus downward.

The fact that there is marked discrepancy between the degree of labyrinthine trauma at operation and the severity of postoperative vertigo, etc., suggests to Jakobi [145] the advisability of testing preoperatively the sensitivity of the vegetative nerve system. It is suggested that patients should be classified as "vegetative stables" and "vegetative labiles." In a series of 66 fenestration patients it was found that the degree and duration of postoperative disturbances of the balance mechanism varied in accordance with the group to which the individual patients belonged and that strong reaction to epinephrine stimulation entailed severe postoperative disturbances of balance, while marked reaction to the injection of adrenergic substances was followed postoperatively by either objective or subjective disturbances of long duration or by marked vertigo without actual imbalance. Sympathetic hypertonicity being most pronounced at pubescence and the climacteric, it is only to be expected that female patients fenestrated at these phases of life should show marked postoperative disturbances of the balance mechanism. Accordingly a preoperative vegetative test is advocated by means of the adrenergic drugs, as affording valuable prognostic information regarding the balance mechanism, and it is advised that, even if the prognosis in regard to hearing is good, in the event the balance prognosis is poor, operation should be avoided, and special caution is essential in the case of patients at pubescence and the climacteric.

The underlying cause of diplacusis following fenestration is a change in the elasticity of the basilar membrane and the

caliber of the canal, with the result that the precise point at which resonance occurs in the basilar membrane is displaced for the individual frequencies either basally or toward the helicotrema, and accordingly a state of dissonance occurs. Various writers have commented on the fact that tones in the fenestrated ear are apparently lower than those on the opposite side. Rossberg,[239] however, found that two otosclerotics who had undergone fenestration reported that tones in the treated ear seemed to be of a higher pitch than those of the opposite side, and an exhaustive investigation was accordingly carried out. The results of these investigations bear a noted similarity to those obtained by von Békésy with preparations and models of the basilar membrane. If, therefore, during the development of otosclerosis altered intralabyrinthine pressure should effect an alteration in the resilience of the basilar membrane, then on fenestrating the ear these conditions are restored to nearer the normal by operation, and accordingly in the fenestrated ear there would be a lowering of the threshold for hearing at each frequency, and at the same time individual frequencies would be heard at a higher pitch on the operative side. It is significant that some otosclerotics who were professional musicians were questioned as to any disturbance of hearing other than that occasioned by swelling. The result of this questioning showed that they had tuned their string instruments too high while they were in the advanced stage of otosclerosis. To summarize, therefore, postoperative diplacusis in otosclerotics may be regarded as being caused by the restoration of the intralabyrinthine pressure on the side of operation to normal.

In 25% of the successful cases after fenestration there is an improvement in hearing by bone conduction in the ear which has not been surgically explored, report Wild, Klotz, and Boeglin.[295] There is also an improvement in hearing by air conduction, which is less. The improvement is noticed toward the end of the first month after operation, and rises to a maximum during the third month; it begins to disappear after six months, and has gone completely after an interval of a year.

The reduction of the initial improvement in hearing after fenestration, which is observed between the fourth and ninth postoperative months, is ascribed by Albrecht [2] to loss of tension of the tympanic membrane. In such cases, bone conduction is unimpaired. Eustachian insufflation results in temporary improvement. A wet swab on the tympanic membrane also helps the hearing. As an experiment, the tympanic membrane was left in its bony frame, and Sourdille's normal technique of fenestration with flap surgery was carried out in the usual way. This was technically possible. No loss of tension has been observed, although the case is too recent to draw definite conclusions.

Shambaugh [262] is of the opinion that after fenestration the patient hears through the fistula in the horizontal semicircular canal. In order to increase the difference in sound pressure between the new window and the round window, Aquaphor (a nonirritating ointment) is applied to the tympanic membrane. The result is that in 45% hearing was improved by 5 db. or more. The total number of patients was 274, the average improvement for the speech zone being 3.74 db. The greatest was obtained in the lower tones, 128, 256, and 512 cps.

Hearing behavior during the first postoperative period of labyrinthine fenestration was examined by Zanotti.[303] On the basis of audiometric, clinical, and histological findings the concept of labyrinthitis following fenestration should be modified. The trauma of a correctly performed operation produces an irritative stimulus which affects only the posterior labyrinth, while there appears to be no sign of extension to the anterior labyrinth. Impairment of hearing for high frequencies from the 3d to 20th day is not related to cochlear damage, but depends on mechanical factors due to an increase of mass of the new vibrating system owing to reactive edema of the tympanomeatal flap.

Quantitative studies of suprathreshold cochlear microphonics in experimental labyrinthine deformations were made with bone- and air-conducted sound by Goodhill, Holcomb, Rehman, and Brockman.[113] Bone conduction deficits of 25 db. may be attributed entirely to labyrinthine occlusions.

Deafness

Congenital deafness is defined by van Egmond [71] as a more or less pronounced loss of hearing, present at birth, leading to mutism and transmitted by heredity or acquired by chemical, physical, or mechanical influences in the prenatal period. In the acquired form the pair of genes alloted for the development of the organ of hearing has undergone a change in composition (mutation). There are two types of congenital hereditary deafness: (1) dominant hereditary, (2) recessive sporadic. Dominant inner-ear deafness is rarely congenital. It develops in middle age, has a strong progression with frequent lengthy pauses, and a loss of hearing for all frequencies. Congenital sporadic recessive deafness appears without known cause in only one child in a seemingly healthy family. If consanguinity exists, the chance of deafness rises. Recessive deafness may be combined with other disorders—disturbed vestibular function, retinitis pigmentosa, feeblemindedness, and hereditary ataxia. These solitary cases of deafness may not be inherited but may be acquired during pregnancy and sometimes also shortly after birth by a nonrecognized infectious disease or other factors before the child is talking. The number of cases of congenital deafness by heredity is presumably half to one-third of all the deaf, recessive deafness strongly predominating. Another malformation that may produce congenital deafness, if both sides are affected, is atresia of the external meatus, often combined with deformed auricles and chiefly with changes in the middle ear, though sometimes the inner ear also may be damaged. This appears to be a dominant hereditary defect, but in most cases an intrauterine acquired development is probable. Erythroblastosis fetalis may also cause congenital deafness as a result of intravital hemolysis caused by an antibody-antigen reaction in the newborn evoked by the antagonism of the different RH factors in the blood of the mother and child. Icterus gravis and evidence of damage to the central nervous system appears (athetosis, rigidity, mental defect, and deafness if the nuclei of the cochlear pathways are involved). During the first trimester of pregnancy exogenous influences such as rubella may give rise to the defects. Syphilis may be a cause of congenital nerve deafness, though the onset of deafness appears at the age of 8-13 years. Extreme caution must be used in administering drugs to an expectant mother, for drugs such as quinine, salicylates, and alcohol have a deleterious effect on the hearing organs of the fetus. Prevention of congenital deafness of the hereditary type is directed toward sterilization with the consent of the subject and prevention of marriage between deaf persons and of consanguineous marriages.

The incidence of various prenatal and postnatal causes of deafness in children under 5 years of age, based on observations made in 270 consecutive cases, are reported by Fowler and Basek.[96] Prenatal causes in 81 cases were hereditary deafness (7), retinitis pigmentosa (1), syphilis (2), Rubella (12), anomalous growth and malformations (7), Rh factor (12), emotional causes (7), vaccinations (6), eclampsia (4), birth trauma (13), infections in utero (3), excessive use of drugs (1), and cyanosis after birth (4). Postnatal causes (189 children) were chronic catarrhal otitis media (26), acute purulent otitis media (10), chronic purulent otitis media (6), cholesteatoma (3), meningitis (10), mumps (8), measles (14), status febrilis (7), streptomycin (2), dihydrostreptomycin (1), aureomycin (2), aspirin (1), carbon monoxide poisoning (2), cerebral degeneration (4), Ménière's disease (3), leukemia (1), and head injury (3). In 85 children the cause of deafness was unknown.

An etiological study based on examination of 111 children 6-15 years of age with high-tone deafness is reported by Johnsen.[147] Hearing was equal in both ears and three-fourths of all the children were able to hear a spoken voice at a distance of more than 6 meters. None of the children were absolutely deaf. Detailed histories were obtained on the prenatal period, the delivery and development in every case, and also details concerning the hearing of several relatives, of which 247 were otologically examined. The cases were divided into three sections: hearing impairment due to (1) endogenous factors; (2) certain environmental factors before, during, and following birth; (3) environmental factors during childhood. Fifty-five families were examined objectively, and pedigrees illustrate three groups: (1) simple dominant heredity; (2) irregular dominant or recessive heredity; (3) cases whose parents are blood relations. Perceptive deafness is of genetic origin in about 40%. In about half of non-hereditary cases exogenous factors were demonstrated. Symmetrical perceptive deafness is often acquired during or immediately after birth. The lesion was due to erythroblastosis in 5%, paranatal asphyxia in 10%, prematurity without demonstrable asphyctic signs in 10%. In one-third of the cases the cause was unknown. Maternal rubella or congenital syphilis were not encountered. Symmetrical perceptive deafness is rarely acquired in childhood. Cerebrospinal meningitis, cranial injuries, and children's diseases were of no etiological significance.

The principal classifications of hereditary deafness are concisely reviewed by De Vido and De Stefani.[64] A report is given of 10 cases in young subjects affected with a particular form of deafness, which was doubtless familial and was proved to involve the nervous structure of the internal ear. Audiometric and vestibular examination was carried out. On the basis of the anamnesis, it could be excluded that the deafness was acquired either during fetal life or later; several individuals in one family exhibited the same type of hyacusis, and it was found that the auditory impairment was always bilateral (a sure sign of the hereditary nature of the affection). All these facts render the diagnosis of hereditary hypoacusis with recessive character reasonably certain.

In an extensive monograph Secretan[257] describes the results of his investigation into a mountain tribe with a very great percentage of recessive deaf-mutism. Many of the examined deaf-mutes showed not inconsiderable hearing rests. Therefore dumbness does not always complicate deafness. This congenital deafness is not always symmetrical. The hearing rests are especially found for the bass, but sometimes a U-shaped audiogram is found. Recruitment is often found. Vestibular function is often abnormal and the more so when hearing is bad. A mental debility is mostly present. Though heterozygotes seem to be free from deaf-mutism, the author found small deviations of hearing in about 50% of the whole tribe. It seems, he states, that the dominance of the normal gene is not absolute.

Congenital deafness and hemolytic disease of the newborn is discussed by Fisch and Osborn.[88] Of 69 cases, 27 showed strong evidence suggesting hemolytic disease of the newborn. The average age of the patients was between 8 and 9 years; all cases had a history of jaundice in early infancy; 10 were anemic at birth, and 6 others had to be given transfusions. Otological examination revealed no evidence of middle-ear disease; vestibular response was investigated in 17 cases by Bárány's turning test, the response being normal in 11 and diminished in 5. In one child a coarse nystagmus with severe vertigo was elicited. There was no evidence of mental retardation in 25 cases, although all children started to talk late and 21 had motor defects. The speech defects were typical of those found in children with perceptive deafness in one group and in another due to both deafness and neuromuscular incoordination. Perceptive deafness of varying degree was diagnosed in all 27 cases, the deafness being severest in the cases with marked neurological complications. In the other 42 cases

hemolytic disease was regarded as possible. A similarity was observed between the audiograms of these cases and those of congenital deafness in cases of anoxia, suggesting a common localization of the lesion if not a common etiology. There is no relationship between prematurity and the degree of deafness. It is suggested that congenital perceptive deafness may be cochlear or nuclear, the latter being the more likely to occur, as the nature of the condition indicates a certain susceptibility to damage of the high-frequency pathway.

The Rhesus factor in deafness is presented by Cavanagh.[37] Among 118 deaf children, 3 cases of Rhesus sensitization were found. Among 124 Rhesus babies, 6 cases of deafness were found, none of which had been suspected. If every Rh-negative woman is delivered where there are facilities for testing the cord blood and for an early exchange transfusion, then Rhesus children should cease to be crippled by kernicterus or deafness. In the meantime, Rhesus sensitization must be considered among the possible causes of deafness. The giving of Rhesus-incompatible blood is doubly dangerous to the female. In an emergency transfusion, if it is not possible to determine the Rhesus group of the patient, we should use Rh-negative blood.

The pathology of rubella deafness is reviewed by Lindsay and Harrison.[174] Rubella in the first trimester produces in the child a sacculocochlear type of malformation of the inner ear. The changes in the cochlear duct appear to be secondary to changes in the stria vascularis with a resulting collapse of the cochlear duct and saccule, indicating a lack or even absence of endolymph. Interference with function may be accounted for by the incomplete development of Corti's organ in some areas and by the pathological state of the tectorial membrane. In spite of lack of development of the pillar and hair cells in rubella deafness, no abnormality has been shown to occur in the peripheral cochlear neuron. The virus reaches the developing sensory structures by way of the blood vessels and endolymph. Saccular changes suggest that collapse of the saccule may have occurred during the maternal rubella infection.

Inner-ear pathology due to measles is reported by Lindsay and Hemenway.[175] Several surveys of deaf children have indicated the frequency of deafness due to measles. Only three previous descriptions of the inner ear in such cases were found. Pathological changes in the ears from a case of measles illustrate the direct effect of the virus on the sensory structures. The lesions affected primarily the stria vascularis. Degeneration of the tectorial membrane and hair cells had occurred throughout. The supporting elements in Corti's organ had disappeared in the lower middle and basal coils accompanied by degeneration of the nerve fibers and ganglion cells as well as the disappearance of the stria vascularis. In the vestibular labyrinth small proliferating granulomatous lesions were present in the wall of the utricle as well as in the macules of the utricle and saccule and in the cristae with secondary degeneration of the sensory cells. The pathological process may be termed endolymphatic labyrinthitis with secondary nerve degeneration.

Lancaster [164] points out the difference in epidemiology between rubella in Australia and in English cities and concludes that there is no need to postulate a difference in the responsible virus in Australia; the scattered epidemics in the less populated areas such as Australia and New Zealand are likely in the future, with an increase in population and travel facilities, to become more endemic in character and thus resemble more the disease pattern as seen in England.

Fleischer [89] reports on the histological aspects of the petrosal bone in a deaf-mute in whose ancestry or offspring deafness does not occur. Caloric testing carried out during life revealed bilateral unexcitability of the vestibular apparatus. A marked malformation of the internal ear was found, with a nearly complete defect of the modiolus and widening of the lateral semicircular canal. It is contended that the developmental

disturbances occurred during the second embryonic month at the latest. At this time, the formation of the system of semicircular canals is nearly complete, although the cochlea has only 1-1.5 convolutions. Data on various embryopathies and experimental investigations suggest that this period represents a particularly vulnerable phase in the organic evolution of the ear. A description is given of two other malformations similar to the first, in an anencephalic and microcephalic fetus, which confirm the views on the point of termination of the malformation. The etiology is discussed, and the possibility of an exogenous injury inflicted in utero is pointed out.

In 1952, 0.7% of the children in Denmark (from the 7th to the 16th year) had so great a hearing loss that the language could not be acquired in the usual way, reports Arnvig.[11] The causes are hereditary hearing defects, 28.5%; hearing defects acquired before birth, 8.7%; hearing defects acquired at birth, 9.7%; hearing defects acquired after birth, 31.6%; hearing defects of unknown etiology, 21.5%.

Hardness of hearing in 2,550 Copenhagen school children was studied by Falbe-Hansen.[77] Hearing defects were found in 295. In 21.7% of these cases the hearing loss was perceptive and in 78.3% the defect was in sound conduction. In the latter group, cure or improvement was attained in 90%. Schools situated in socially poor sections had 15.5% pupils with hearing defects, schools in favorable sections only 9.7%.

Johnsen[146] investigated 111 school children with partial perceptive deafness, none of whom attended schools for the deaf. These children had considerable impaired hearing for the whispered voice, but much less so for the spoken voice. Audiograms fall into three groups: (1) with abruptly descending curves, (2) with more gradually descending curves, and (3) with a hearing loss over the entire range. In the first two groups hearing of the low tones is normal. Within each group there was no demonstrable difference between the ears with air or bone conduction and no difference between the sexes or age groups. The lesion, then, is not progressive during school age. Nearly all cases showed normal vestibular responses. About half the children had speech defects consisting chiefly in omissions and substitutions of consonants. Thirty-five of the children had not uttered words until the age of two years.

A group of 60 children suspected of having impaired hearing because of speech deficiencies were studied by McHugh and McCoy.[191] The average age was 31 months. Multiple handicaps were present in 42%. Insufficient hearing to learn speech occurred in 30 children. Of these, 14 were deaf; 14 had a very severe hearing loss (80 db. or more), and 2 had a severe hearing loss (60-80 db.). The remaining 30 failed to develop normal speech because of brain injury (17 cases), mental retardation (8 cases), and severe emotional disturbances (5 cases).

The belief persists in many quarters that the inarticulate child must be investigated by elaborate laboratory methods. Puretone audiometry, PGSR audiometry, peep shows, and electroencephalography are not suitable for diagnosis of speech and hearing defects in young children, reports McLaurin.[192] Instead, diagnosis is accomplished, and in this order, by detailed history-taking, clinical observation, and clinical evaluation, with laboratory investigation last of all. The use of noise makers in a free field, by the so-called cessation of activity technique, is a desirable method of testing hearing in young children because it can be done unobtrusively, in natural circumstances.

The typical decrease of the eosinophil count in the morning between 6:30 and 9:30 was investigated in a group of 35 deaf children (5-13 years) by Feldman, Feinmesser, and Landau[81] and compared with an equal group of normal children under the same conditions. In the first-mentioned group, the eosinophil count was reduced by an average of 8%, in the normal children by an average of 29.9%. The conclusion is reached that sound stimuli play an important

role in the causation of a decrease in the eosinophil count in the morning (the sound stimuli probably activate the hypothalamic centers, from which the stimulus is transferred to the hypophysis, which produces ACTH).

Two main divisions of deafness, congenital and acquired, are reviewed by Campos de Henriques.[59] Congenital deafness may be either biological or pathological, the first comprising heredity and fetal erythroblastosis, and the second comprising intrauterine infections, endocrine alterations, and intrauterine intoxications. Acquired deafness may have several causes, viz., trauma, intoxication (either exogenous or endogenous), infection (bacterial and viral), and nasopharyngeal causes. In true deaf-mutism the lack of speech is always secondary to deafness, provided this is severe enough and provided it be present at birth or acquired before 6 years of age. Heredity: In the human cochlea one may find three different histological types of lesions corresponding to the well-known Michel's type, Mondini's type, and Scheibe's type. The above-mentioned morphological consequences of heredity result from Mendel's laws. Prophylaxis is based on eugenic measures. Fetal erythroblastosis: The general ideas and the pathology of kernicterus are reviewed. Prophylaxis is tried by means of premature delivery at the seventh month of pregnancy, exsanguino-transfusion, and administration of cortisone to the pregnant woman. Intrauterine infections: Syphilis and its pathological consequences are discussed. Viral infections of the mother during the first four months of pregnancy, with particular reference to rubella, poliomyelitis, and infectious hepatitis, are reviewed. The similarity of viruses and genes is remembered in order to understand the similarity of the lesions found in the cochlea either in Gregg's triad or in Scheibe's congenital type. Prophylactic measures usually fail in viruses. Endocrine alterations: Thyroid deficiency, as seen in endemic cretinism, in congenital athyreosis, in Van der Hoeve-De Kleyn syndrome, and in Laurence-Moon-Biedl syndrome, is given full consideration. Intrauterine intoxications: All the toxic conditions which may be called classic are remembered, as well as their mode of action on the hearing organ; special attention is given to streptomycin, particularly to dehydrostreptomycin. Prophylaxis is obvious. Trauma: It is not a common cause either during delivery or after birth. Intoxications: exogenous intoxications are the same both for congenital and for acquired deafness. Two main endogenous intoxications are recalled: postpartum anoxia and nephritic labyrinthitis. Progress in therapy is the best available prophylaxis. Infections after birth: Viral and bacterial diseases which may lead to deafness are briefly reviewed and their prophylaxis remembered. Nasopharyngeal causes: They very rarely produce deaf-mutism.

Neuberger[208] considers the threshold audiogram in cases of labyrinthine (perceptive) deafness not only as a series of physical data about the hearing function at a certain moment, but also as the expression of a phylogenetic principle. The receptors for higher frequencies are supposed to be phylogenetically younger than those for lower frequencies and thus more vulnerable. The vestibular is the phylogenetically eldest and thus least vulnerable function of the labyrinth. Ontogenetically the several parts mentioned develop in the same succession: vestibular function, low-tone perception, high-tone perception. These ideas are demonstrated in cases of congenital deafness, among which the author recognized two types: I, with more or less steep decline of the audiometric curve from 512 to 256 cps upward; II, with hearing loss more pronounced in lower than in higher tone areas. By noxae acting during intrauterine life the phylogenetically and ontogenetically younger parts are damaged before the more resistant older parts. Only heavy and early acting noxae can extinguish the vestibular function in intrauterine life. The author considers the intralabyrinthine vestibular organ as the matrix for audition. Audition cannot develop when the vestibular organ is ab-

sent. In cases of both types the vestibular function is always present when there are hearing remnants. In the families of patients of Type II there is always incidence of congenital deafness. This type must be ascribed to hereditary factors, which do not interfere with the development of the receptors for high frequencies in later intrauterine life. During extrauterine life labyrinthine deafness by endogenous causes and by chronic acoustical trauma (occupational deafness) follows the same rule: the phylogenetically younger elements are damaged before the older elements.

Lehmann [171] reviews the literature on bilateral sudden deafness and reports two cases of his own. Possible causes of sudden deafness include intralabyrinthine hemorrhage due to arteriosclerosis or blood dyscrasia; lesions of the central nervous system; toxic neuritis; late syphilis; angiospasm of the vessels supplying the labyrinth; perilymphatic hemorrhage following trauma without fracture. Psychogenic deafness must also be considered. In the first case a 28-year-old male Negro, previously treated for syphilis, became suddenly deaf in both ears. Massive penicillin treatment was started within 24 hours, and both ears recovered completely. The other case was a 49-year-old white man who, the night before examination, had fallen on his face on the hospital grounds; hearing was completely lost on the left, and the patient was profoundly deaf on the right, particularly for high tones. The author feels that this is a case of psychogenic deafness, because hearing in the right ear showed some fluctuations. Since there was no recovery during an entire year, traumatic hemorrhage into the perilymphatic space must also be considered.

The hearing of 300 diabetic patients was carefully examined audiometrically by Schröder.[253] In no case was a disturbance of hearing attributable to diabetes found.

The concept that nerve deafness must be progressive is no longer tenable, according to Juers.[151] Therapeutic failures in the past have resulted from attempting to reverse advanced cochlear pathology and relying solely on the use of drugs whose pharmacological effect on the ear was uncertain. Early end-organ deafness may be reversible. Smoking may be occasionally a cause, also psychological stress, excessive use of caffeine, allergy, and endocrine imbalance. Thus a broad physiological approach to a wide range of etiological factors is perhaps the most important aspect of the general problem of early nerve deafness.

Syphilitic disease of the inner ear, reports Wageman,[286] has greatly diminished during the last 15 years. The diagnosis cannot be made from hearing tests alone. Usually vestibular disturbances are also present. Once it was thought that excessively short bone conduction is indicative of syphilitic disease, but this assumption was not borne out. Severe cochlear and nerve deafness in the younger age group should arouse suspicion of syphilitic disease. Often the low frequencies are affected, and, as in other types of cochlear damage, recruitment will be present. In cases responding to treatment Zangemeister demonstrated early reversal of recruitment.

The Cogan syndrome, described by Cogan in 1945, is characterized by sudden photophobia, ocular pain, blurred vision, and blepharospasm. Within a few hours to a few weeks tinnitus, bilateral progressive deafness, vertigo, and vomiting develop. Interstitial keratitis is an early finding. Serological and clinical examinations demonstrate the absence of syphilis. Eosinophilia is usually present. Sometimes the ocular symptoms predominate, at other times the eighth nerve symptoms. The disease is bilateral; the cause is uncertain. For therapy, convalescent plasma, histamine, vitamins, cortisone, and cervical sympathectomy have been used, all without success. Stevens [268] reports a case of Cogan's syndrome, the first one in a child. Symptoms developed over a number of years, and no therapeutic measures could arrest the progress of the disease.

Robinson [232] points out that it was in 1905 that Vogt described a nontraumatic uveitis associated with alopecia and graying

of the eyelashes and hair, the condition now known as the Vogt-Koyanagi syndrome (uveitis, deafness, alopecia, and loss of pigment in the skin and hair of the scalp, the eyebrows and eyelashes). It is in a way akin to Haranda's disease, in which there is a low-grade uveitis associated with bilateral detachment of the retina, deafness, alopecia, and pigmentary changes. A case of this relatively unusual syndrome is described, with a report of the full investigations carried out.

Simpson [263] reports and analyzes three cases of aphonia and deafness in middle-aged women, and traces them convincingly to hyperparathyroidism. In this disease motor symptoms are alarming to the patient, but sensory symptoms other than pain tend to be ignored by both patient and examining physician. The auditory symptoms, ranging from singing in the ears and tinnitus to severe deafness, disappeared in all three instances with removal of a parathyroid adenoma and return of the biochemical picture to normal. Concomitant aphonia in one of these patients ceased simultaneously. All three cases presented the classical symptoms of hyperparathyroidism. In the case associated with aphonia, increased nerve accommodation was demonstrated, which is known to result in reduced response of a nerve to a stimulus. The tendency to spontaneous remission shows that deafness was due to biological-chemical disturbances, not to bone changes or metastatic calcification. Chemical deafness is rare, but should increasingly be taken into consideration in patients with a markedly elevated blood calcium level.

Fickentscher [84] reports a case of bilateral, severe, perceptive deafness, together with a review of the Klippel-Feil syndrome and its variants. The 50-year-old patient presented synostosis of almost all cervical vertebrae. External auditory meatus, middle ear, and Eustachian tubes normal; pneumatization on the left good, on the right impaired. Audiometric tests demonstrated a high degree of interference with sound perception, indicating ganglionic or nerve deafness. The audiometric curve remained the same upon change of body position. The Klippel-Feil syndrome was first described in 1912. It consists of fusion of the bodies of the cervical vertebrae, combined with a very short neck and a hairline going down to the back. Spina bifida is frequently present, together with other congenital anomalies. Elaborating on the differential diagnosis the author feels that this case, with good vestibular function, constitutes recessively inherited congenital perceptive deafness. Fusion of the cervical vertebrae may also be acquired, as in osteoporosis; there may be hearing loss, but it is usually unilateral and not as severe as in congenital cases; on the other hand, vestibular symptoms are often present.

Ear disease associated with deformities of the cranial bones is frequently encountered, and is mostly due to inflammatory changes or tumors; however, systemic bone disease affecting hearing, particularly osteitis deformans, is rare. Gollmitz [114] reports two cases. The first one presented the typical picture of osteitis deformans with unilateral bulging of the right half of the cranium; in the second case blood chemistry and skeletal x-ray study had to be performed before the diagnostic problem could be resolved. Deafness is mainly traced to interference with sound transmission; if perceptive, the disturbance of inner-ear hearing is due to direct obstruction by bony deformities. Suppuration of the middle ear is fomented by the bone disease; it is explained on the basis of a physiological disturbance due to narrowing of bony structures with resulting interference with aeration and blood supply. In the treatment, hormones and vitamins as well as irradiation were tried without any success.

A case of deafness and cleidocranial dysostosis is reported by Davis.[57] The disease may affect the entire skeleton to a greater or less degree. It is a congenital abnormality involving the bones of membranous origin. The pathognomonic features are found in the skull, clavicle, and pelvis. The eyes are widely spaced (hypertelorism);

the nose is depressed at the bridge; the face is small, and the teeth show faulty eruption.

The incidence of inner-ear deafness of unknown etiology is discussed by Gerlach.[105] Symptoms of disturbance in liquor circulation were found in many of these cases. The main causes are thought to be due to a past meningopathia adhesiva or some anomalies of the skull. Symptoms referable to the cochlea are common, while vestibular symptoms are secondary. X-ray of the skull and audiograms are shown to demonstrate an improvement of hearing after drug treatment (dehydration, changes in liquor pressure) or in cases of anomalies of the skull by performance of a decompressive operation.

Lees and Munro [170] draw attention to the comparatively infrequent symptoms in disseminated tuberculosis. Eight cases are presented in which cutaneous lesions were the only reliable lead to correct diagnosis and prompt treatment, which is the prerequisite of success. In three of these cases hearing was involved to varying degrees. In one of these the patient became moderately deaf, and in addition there were little areas of faucial ulceration, postnasal discharge tinged with blood, and bright red petechiae behind the ears. In another case hearing deteriorated over a period of two months, and at time of admission the patient was almost totally deaf. The very first symptom was dysphagia, without any signs of throat involvement. Subsequently hoarseness and pain in the throat developed, and at time of admission, tongue, uvula, and pharynx were inflamed and ulcerated. The third patient became moderately deaf, but physical examination was almost completely negative. The authors conclude that deafness may be of some diagnostic importance in certain cases of acute tuberculosis. Occasionally tuberculosis leads to severe deafness which disappears with successful treatment of the underlying lung disease. In most of the cases they reported the diagnosis of tuberculosis was made by microscopic examination of biopsy specimens from the skin lesions.

Neurinomas rank third in tumors of the brain; they are usually unilateral, and histologically they are schwannomas which have developed in the peripheral portion of the acoustic nerve. The onset, according to Geraud et al.,[104] is cochleovestibular, the initial symptoms being auditory symptoms; tinnitus on the affected side, which may occasionally persist for years on end, and a progressive unilateral impairment of hearing, with perceptive deafness. Vestibular symptoms are marked by an early onset usually accompanied by deafness; vertigo is diffuse, although it occasionally simulates Ménière's syndrome; the horizontal rotatory nystagmus of labyrinthitis is a pure horizontal nystagmus with an occasional vertical component; spontaneous displacement of the index is unilateral, and Romberg's sign is observed on the affected side; the caloric test shows a diminished excitability or total absence of excitability; at this time there are no subjective changes of the trigeminal nerve, but there frequently is a corneal hypoesthesia. In the acute stage there is a tendency to lateropulsion when walking; examination reveals a central vestibular syndrome. The trigeminal nerve shows marked changes; there are paresthesias and formication and a burning sensation in the corresponding half of the face; a facial neuralgia may be the most prominent symptom and the corneal reflex is abolished in the majority of cases. The facial nerve shows slight, occasionally transient, changes; the changes of Wrisberg's intermediary nerve give rise to painful anesthesia. Cerebellar symptoms are frequently observed. The roentgen diagnosis is established by the incidences of Towne, Stenvers, and Hirtz; ventriculography and cisternography may also provide useful data. Treatment should consist in total removal of the tumor.

Early diagnosis of acoustic neuroma is discussed by Elliott and McKissock.[73] Present operative mortality is 10%. Usually the tumor is quite large when diagnosed and operated upon. Diagnosis should be possible when confined to disturbances of the eighth nerve only. The first symptoms is

deafness, tinnitus, or vertigo in 75% of the cases. Twitching of face and light headaches come later. Important early signs: perceptive deafness, reduction of corneal reflex, facial weakness, and unsteadiness of gait. Special x-ray examination of internal auditory meatus, caloric tests, and audiometry. Most important to investigate is the presence of recruitment. Presence of full recruitment is against acoustic neuroma. Reduction of corneal reflex may be due to interference with the facial nerve.

Repeated cochlear and vestibular examinations at one-to-two-week intervals show no changes in compact tumors of the posterior cranial fossa, according to Hibler.[130] If arachnoid processes are present, there is a rapid loss of hearing. Cystic processes cause a marked fluctuation of the vestibular reactions. Three cases are reported with slight headaches, vertigo, tinnitus, and unilateral progressive hearing loss. Vestibular responses were at first hyperactive, then subnormal. Exploration showed marked stasis of cerebral fluid in the lateral cistern with normal finding otherwise. After the surgery, hearing returned nearly to normal. No recurrence was noted. Of 140 brain tumors reported, 69 had normal cochlear and vestibular findings. Tumors of the parietal and parieto-occipital lobes showed loss of hearing on the side of the tumor in 70% and a vestibular hyper-excitability on the other side due to pressure from distorted ventricles.

Wildhagen[296] examined 120 cases of brain tumors audiologically; 75% showed disorders of hearing. Eighth-nerve tumors are characterized by homolateral deafness. All other tumors in the various parts of the brain produced a symmetrical hearing loss of perceptive type with a progressive loss from the low to the high tones. Thus, deafness due to brain tumor is a symptom of increased intracranial pressure. Like the elevated papilla, it cannot be used for a localized diagnosis. The author could not confirm the typical findings of crossed and asymmetrical deafness of the midbrain origin.

With the aim of bringing to evidence the "hidden" auditory loss in temporal lobe tumors, a method of speech audiometry was devised by Bocca, Calearo, and Cassinari,[25] using distorted instead of normal voice. The articulation score is poorer in the ear contralateral to the side of the tumor. This seems to warrant the assumption that when the auditory cortex is involved by the growth the psychic integration of the verbal message may be more or less impaired.

It is important to distinguish between two forms of deafness following acute pyogenic meningitis, state Maspetiol, Pigement, and Tronche.[183] Fifteen patients were studied. In seven there was evidence of nerve-deafness and this was permanent and irreparable. In eight patients the deafness was of cochlear origin. In these some improvement of hearing is possible. In tuberculous meningitis deafness is almost always due to the streptomycin, but it may also be due to the meningitis, as it is in other forms of meningitis. A method of differentiating between cochlear and nerve deafness is described. Treatment for cochlear deafness with dietary restrictions and drugs is suggested.

The Ramsay Hunt syndrome is classified into four clinical types: (1) herpes oticus with no neurological signs; (2) herpes oticus with facial palsy; (3) herpes oticus with facial palsy and auditory symptoms; (4) herpes oticus with facial palsy, with accompanying auditory and labyrinthine symptoms. The 12 cases presented by Harrison et al.[127] were unilateral; all occurred between the months of February and September, and they were all in adults. Only 10 could be traced and of these 5 showed complete recovery of the facial paralysis. Of the cases which showed perceptive deafness, only half of them had improvement in the hearing to normal limits. Zoster infection is not confined to nerve ganglia and there is clinical and pathological evidence that it may cause neuritis of the motor nerves. Thus, it is not necessary to postulate that invasion of the ganglion actually occurs when facial palsy is associated with herpes

of the head and neck. The term "Ramsay Hunt syndrome" should be used only for those cases of facial palsy in which the herpes is restricted to the conchal zone. A brief account of three cases is presented, with the results of audiometric and caloric tests. Only two of the cases had herpes but all had facial paralysis.

Keutner [156] established that, in contrast to what had been assumed before, the cochlear nerve is frequently affected by multiple sclerosis, but the hearing impairment often remains unknown to the patient and can be detected only by audiometric tests. Fifty patients with neurologically demonstrable multiple sclerosis were studied. This series did not include any patients in whom deafness could be traced to ear disease like chronic catarrh or past or present suppuration, and no subjects over 50 years of age were included in the group. In 46 cases audiometry revealed a hearing deficit mainly in the low and high frequencies. Thirty-nine of these subjects were not aware of any hearing impairment. Air-conduction as well as bone-conduction audiograms show dome-shaped curves, with no loss in the 512-2,048 frequencies. At either extreme end of the tone range, the loss may be as high as 70 db. The dome-shaped curve of the audiogram indicates concentric narrowing of hearing, which points to central deafness due to involvement of the brain stem. This is well in accord with the fact that multiple sclerosis is often traceable to changes in the brain stem. As long as the speech frequencies are not involved, the patient is not aware of a hearing impairment, nor can it be detected by any other procedure than the audiometric test.

Otorhinolaryngological examination of 79 "Ama"—diving fishermen and women—was made by Yoshio Kataoka.[301] The more years they have continued to work and the deeper they dived, the higher was the degree of their hearing loss. No abnormality of the vestibular function was found.

A study was made by Salis and Chessa-Perle [247] of a group of subjects who were blind either from birth or from early youth, in order to determine whether the loss of vision had been of influence on the acuity of hearing and of various forms of corporeal sensitivity. The study was inspired by the finding that there exists interdependence between the physiopathology of vision and that of hearing and by the fact that many associative neurons connect the general sensitivity. The study material was formed by a group of 20 persons, of both sexes, ranging in age from 12 to 36. It was found that only one subject among the 20 examined presented normal hearing acuity, all the others showing hypacusia of pantonal type. Surface sensitivity was found to be less than normal, as were paraesthesia and osteo-articular sensitivity. Further findings were a discordance between deep and superficial epicritic sensibility, and a deficient special sense. In conclusion, the author confirms the validity of the concept of functional synergism of the various organs of sense, leading to a perfectly harmonious function of the various senses.

Wise suggestions in the management of deafness are offered by Day.[58] If medical or surgical treatment is ineffective, do not continue to treat your deafened patients or give them hope that their hearing may improve. They will make no attempt to adjust to their handicap so long as they have any hope to improve hearing from treatment. Never tell them that their hearing will get worse. Explain the use and limitations of hearing aids and have them get instruction in the use of hearing aid and in speech and auditory training when indicated. Deafness should be accepted as an incontrovertible fact and determination acquired to overcome the disability that it causes. A hearing aid should be procured and worn continuously. Be attentive and use your eyes and brain to help you listen. Receivers should be worn in both ears if discrimination is improved, even though this entails the added problem of learning to control the modulation of the voice. Avoid situations where acute hearing is essential. Avoid the antisocial tendency of the hard

of hearing. Develop a sense of humor about your deafness.

Among the indications for a tonsillectomy and adenoidectomy, the loss of hearing is the most important. There are many incidents of middle-ear effusion without real signs of infection. If this condition is neglected, a partial but permanent hearing loss may ensue. Hoople [140] warns that pediatricians are not always aware of what might happen to the hearing; the use of antiobiotics may prevent the development of infection but remain without effect on effusion. Allergy and hypertrophy of nasopharyngeal lymphoid tissue create a vicious circle. The lymphoid tissues should be removed even in a three-month-old child. In these early cases the tonsils should be spared whenever possible. Early removal is necessary because one can never know whether an attack will result in permanent damage to the hearing. The author advocates adenoidectomy under vision, removing all tissues until the nasopharyngeal fascia is clear. Children who had this operation before the age of 3 years might have to be reoperated on later on, but this does not detract from the great benefits derived from surgery. The author is definitely against radiation, because he never saw lasting good results from it.

Improvement in hearing in cases of perceptive deafness with positive recruitment by use of vitamin A is reported by Rüedi.[242] Apparently hair-cell lesions respond to vitamin A therapy unless there are associated changes in the ductus cochlearis (Ménière's disease and acoustic trauma). The simultaneous use of large doses of vitamin A to some extent inhibits the toxic effect of streptomycin and of neomycin in the inner ear of the guinea pig. During the administration of 100,000 U vitamin A daily an acceleration of adaptation to minimum sound intensity occurs in the human ear (a shortening of physiological fatigue) and the intensity of noise which the test subjects can maximally tolerate without pathological loss of hearing increases significantly by 10-15 db. Vitamin A, then, is effective against acoustic trauma.

Perceptive deafness was treated by vitamin B complex, amino acids, and histamine acid phosphate in 100 cases over a 10-year period by Childrey.[44] There was increased hearing loss of 2.5% binaurally after 9 months, and while treatment did not result in improved hearing, it may have slowed the progress of the deafness although probably not enough to justify the expense and effort expended.

After discussing the results and the probable action mechanism of intramuscular thiamine diphosphate on vestibular and acoustic function, Branca and Germana [30] affirm that no influence is exerted by it on the vestibule, whereas acoustic function is highly intensified.

Nakamura [206] conducted experimental study on cepharanthine therapy of perceptive deafness in guinea pigs injected with BCG in order to prove experimentally the clinical results. According to the results, it may be confirmed that deafness due to tuberculous allergy will recover with early treatment and not recover if the treatment is delayed. The author made clear that deafness due to tuberculous allergy can be arrested through cepharanthine treatment, and that there is a limit to that treatment.

Chladek [46] treated the inner ear hardness of hearing of various degrees and Ménière's disease. He used the method of Harris as a modification of the original histamine infusion according to Horton. The improvement was obtained approximately equally in all patients and frequencies. Immediately after the treatment the gain was smallest, and later, after three to four weeks, the results obtained were more favorable. In 24 patients the average gain for one frequency after stopping infusion was 2.5 db. After three to four weeks the average gain was 9.15 db. for one frequency. The value obtained after seven weeks was an average gain of 8.4 db.; the remaining numbers were only slightly worse than after three to four weeks. The author explains this "negative period" immediately after the

histamine treatment as the result of raised permeability of blood vessels. Therefore, he recently uses 0.25% of procaine instead of physiological solution in drop infusion or the simultaneous application of pilocarpine. The results obtained in this way were still better. The following injection of histamine given subcutaneously is used to elaborate the conditioned reflex. The author uses for this purpose 0.2 ml. 1% histamine and hyaluronidase injected into the planum mastoideum.

Portmann and Grimaldi [220] describe an apparatus to obtain tubotympanic insufflation with pulsating aerosol. Experimental studies on an artificial model, on animals, and on man show that the aerosol cannot penetrated by insufflation beyond the pharyngeal orifice of the Eustachian tube unless the eardrum is perforated. However, if the air is introduced by interrupted positive pressure, the insufflation can make aerosol reach the tympanic cavity. This positive pressure must have the mechanical characteristic of a square wave. Its action is first of all mechanical on the middle ear and internal ear, secondly reflex (neurovegetative), pharmacodynamic (according to the drug used), and perhaps electric (ionic effect according to the physical status of the drug). All deafness should it be of transmission, mixed or of reception, for which there is no more specific treatment, can be considered as an indication for the method. Several observations of different deafnesses are reported with speech, tonal audiometric and accoumetric control. In more than 50% of the cases an improvement is observed.

Observations from a controlled study on the effect of nasopharyngeal irradiation in a group of school age children are reported by Hardy and Bordley.[126] The problems considered in their five-year study were (1) to determine the efficacy of nasopharyngeal irradiation for the reduction of lymphoid tissue; (2) to study the changes that take place in hearing acuity following irradiation; (3) to investigate the effects of puberty on lymphoid growth and on hearing acuity; (4) to determine the efficacy of modern diagnostic techniques for predicting hearing loss in a school population, and (5) to develop procedures for screening a school population and for carrying out a conservation of hearing program which would be feasible for adaptation into a school health program. Their findings show that nasopharyngeal irradiation is a satisfactory method to reduce lymphoid overgrowth around the Eustachian tube orifices. In children suffering from hearing loss of Eustachian tube origin, irradiation of the nasopharynx has an important additive therapeutic effect beyond that related to maturation.

The nasopharyngeal lymphoid tissues may regrow even after repeated removal. Usually infections of the upper respiratory tract stimulate the growth. The lymphoid tissue prevents proper aeration of the middle ear. In a first series, 5,000 children were studied by Bordley.[27] Three treatments were given with radon or with a radium salt applicator, but never in the presence of upper respiratory infection. In 1948-1949 a new series was started: 500 children were selected and half of them served as controls. At the start the average age was 8 years. The two groups were studied for five years and all children examined every six months. A 50 mg. anhydrous radium sulfate applicator was used, screened with 0.3 mm. Monel metal. Three treatments were given two weeks apart. Marked reduction of lymphoid tissue took place after irradiation. No injury or ill effect was noticed. Cases of severe, fluctuating conductive deafness improved satisfactorily. Irradiation proved to be a safe treatment for reducing a small mass of lymphoid tissue.

As the congenitally deaf child has no communication with the outside world, it is one of the most handicapped, report Fry and Whetnall.[99] There are many methods to train and educate deaf children; each of them has commendable aspects, but none can be considered "the best." The oral method is perhaps superior to manual sign language. If residual hearing is present,

training should start with the development of speech. Preliminary to teaching the deaf, it is necessary to understand the development of normal speech and hearing. Normal hearing involves not only the perception of sound, but its meaning must be understood and comprehended. The cortex learns by frequent repetition, until it is able to discriminate. The precursor to cortical comprehension, is the readiness to listen, which must be developed during the first year of life. The readiness to speak develops usually at the age of 12-18 months, followed by the actual use of language. The behavior and speech of the deaf differ according to the age at which deafness was acquired. The later deafness strikes, the better will be the speech. In order to treat deaf children successfully, the same conditions must be created which are necessary for the teaching of normal children. Each child should have an individual hearing aid. Young children like the aid, wear it happily, and are very upset when it is taken away. Older children starting to wear an aid dislike it and resort to every excuse to try to escape using it. As a rule, they have passed the age when sound discrimination is learned, and thus all sounds are unpleasant noises for them. The congenitally deaf child should first learn to hear and later be taught lipreading, as otherwise he will be incapable of listening. Proper environment, individual attention, and special training are important and should begin as early as possible for the above-mentioned reasons, in order to prevent frustration. The normal word-sentence relation will be least difficult to acquire by children who had the advantage of early auditory training, used a hearing aid early, and later attended ordinary public schools.

Vuillemey [284] emphasizes the value of lipreading. The modern concept of lipreading is complex. First of all it is the reading of visible "phonator organs," as tongue, lip, jaw; secondly, it is also the reading of the facial muscle movements; the muscular picture is inseparable from the lip picture. In spite of this the facial alphabet of phonetics presents several blanks. But the lip-facial-reading perception of speech is much better and easier than the lip-facial-reading of isolated phonetic elements. Indeed during speech, phonetically isolated elements are distorted by many new factors (contiguity, duration, intensity, etc.) which help their recognition by the deaf person. So the lip-facial-reading is facilitated by "mental substituting" and by the intelligent interpretation of the visual kinetic picture.

The state of Illinois has established several summer residential centers for the speech and hearing education of handicapped children, reports Kelly.[155] The program consists of day-long diversified activities. Interesting innovations are the following: 1. Theater therapy with structured conversation. The children prepared a show for the end of the semester, and the goal is practice in communication. 2. Music therapy with informal instruments, group singing, and music appreciation. 3. Training exercises in "little language." Little language is simple communication made up of numbers, alphabet letters, and easy words in series but out of context. It is a talker-listener drill designed to develop discrimination. 4. Speech-hearing training. Six children form a class, and they are arranged in three teams in which one is the talker, the other the listener. Two periods of lipreading were given to each child every day. Each child had his own film viewer in which he could see the illustration of the sound study. The see-and-say movie is a great advance in therapy. Progress was evaluated by pre- and post-training tests in speech hearing, lipreading, and speech. The described group method of teaching proved quite satisfactory. Particularly the inclusion of theater and music therapy seems promising.

In 1951 there were about 10,000,000 people with some hearing loss in this country. Of these, 4,000,000 had serious handicaps, and there are today approximately 3,000,000 children with subnormal hearing. Hearing and speech of every child should be tested when he enters school, and the examination should be repeated each year. Still early detection presents great diffi-

culties, according to Fowler[95]; furthermore, there is a shortage of well-staffed hearing centers for the diagnosis, treatment, and management of hearing and speech deficiencies. General awareness and willingness to cooperate will help early detection of hearing disorders and thereby further hearing conservation. Parents should be instructed to watch hearing and the development of speech in their children during the first few years. In school, teachers should be on the lookout for hard of hearing children. The author gives some leads for early detection of hearing deficiencies, and stresses the importance of alertness and cooperation on the part of the parents. Parents should be instructed how to train the deaf child, because good home environment and individual teaching must go hand in hand with early auditory training. Even the totally deaf are helped by a hearing aid. Until the child has become used to the aid he should attend a special school. The author enumerates a number of "don'ts": do not wait for the establishment of ideal hearing centers in your neighborhood; do not delay to report the deafness of your child; do not delay medical care; do not deprive the child of a hearing aid; do not temporize in the false hope that the child might outgrow his predicament. At present, too much is expected of schools and teachers and too little of the parents.

Indications for hearing aids in young deaf subjects are discussed by Maspetiol and Gautie.[182] Preschool education of deaf children should be started as early as possible; the first years of life are those most important for education. Preschool education of deaf children has three objects: (1) to teach lipreading and prepare for the use of speech; (2) to develop the mind by educational games; (3) auditory training based on the presence of hearing rests of varying value in three-quarters of the cases. Training involves the problem whether or not to use hearing-aids. The indications are complicated, and the following factors must be taken into account: (1) the value of the hearing rests, supraliminal hearing, and the ability to discriminate; (2) the mental development and character of the child and the mental level and character of the mother, an extremely important factor in preschool auditory education. Only compression apparatus should be used in cases of distressing lowering of the auditory threshold. This type of hearing aid is to be preferred in deaf children when recruitment is absent.

The hard of hearing infant is discussed by Wishart.[298] A majority become deaf before the age of 2 years, and few are totally deaf. As a rule there is some residual hearing which should be made use of in the education of the child. It is of utmost importance to detect deafness and commence training as early in life as possible. Deafness can be detected in the first year of life. During this time elaborate tests of hearing are unnecessary. The infant can learn to comprehend speech through lipreading by casual and effortless watching of its mother's face. Many deaf infants have achieved articulation readily and also some speech without loss of spontaneity. The methods of examination and training must be adapted to the stage of the child's mental growth.

Objections are raised by Wishart[297] against a number of errors leading to delay in the treatment of deaf children. Training of the hard of hearing child should begin as soon as the deafness is presumed. The mother is the appropriate person to educate her baby. She should teach it lipreading. To this end, a list of words and short sentences (in English) is given, destined for children from 6 to 24 months, which may serve as a basis for the teaching of lipreading. The hearing tests applicable in infants from 6 to 24 months are reviewed. If an infant is diagnosed as deaf, but shows residues of hearing, it should have a hearing aid as soon as it is capable of wearing one, but with certain precautions.

To obtain objective audiometry electroencephalography was used by Appaix and Avierinos.[9] They discuss the importance of this method in deaf-mute children; however the EEG does not constitute a precise

means for testing whether a child is deaf or not. In some cases it might perhaps help to detect the location of the lesion, particularly when "K" wave complexes are found. Tinnitus is more characteristic. Many tinnitus patients show a very high percentage of "neuronic hyperexcitability." Tracings in Ménierè's disease and total deafness show that the responses are not specific at all. In Van der Hoeve or De Kleyn's syndrome mesocephalic deviations should be shown.

Conditioned galvanic skin reflexes were used by Goldstein, Ludwig, and Naunton [111] to test the hearing of 42 deaf children. Two groups were found that differed in the ease with which they were conditioned. In one the children were conditioned after only two or three presentations of the tone-shock combination and, with little or no reinforcement, they continued to respond to audible tone throughout the test. In the other group the children were conditioned, if at all, only after many presentations of the tone-shock combination and they continued to respond for only a very short time, necessitating frequent reinforcement throughout the test. In the majority of the latter group aphasia was suspected beforehand, and in the majority of the former group aphasia was absent. These results imply that threshold audiometry using the conditioned skin response as an indication of hearing may prove to be no more successful than current techniques in the very cases where current techniques are inadequate. The audiograms obtained by this conditioned reflex method were misleading in the cases where conditioning was difficult, in that the PGSR test always suggested poorer hearing than was indicated by conventional tests. The relation between difficulty of conditioning and the evidence of aphasia suggests that the PGSR may serve to confirm a clinical diagnosis of communication disorder other than peripheral deafness.

As the ability of discrimination is very high in early childhood and decreases after the age of 5 to 6 years, it is very important to start auditory training as soon as possible in children severely hard of hearing. The best results, according to Hildernisse,[131] were obtained by means of a loudspeaker. Hard of hearing children who have developed speech (though defective) spontaneously require auditory training, too, but in a different manner. These children have to learn with their hearing aids quite new acoustic patterns, replacing the distorted patterns they have recorded thus far. This requires teamwork among audiological, speech therapeutical, phonetical, and psychological specialists. Lipreading is also a very important factor. The best results may be obtained in special schools for hard of hearing children. Auditory training in cases of streptomycin deafness in children proved to be ineffective in most cases.

Holmgren [137] counsels that in the selection of hearing aids medical, technical, educational, social and psychological factors will have to be considered. Before ordering an aid, audiometric evaluation will give a hint at what can be expected. The speech-hearing test is of eminent importance. Testing of children is thoroughly discussed, particularly the peep show or toy audiometry. For the 1-2-year age group, the psychogalvanic skin resistance test has proved very satisfactory. No effort should be spared to find out all circumstances related to deafness. Deafness acquired in early childhood, late childhood, adolescence, adult life, and in the aged contributes different problems, and psychological factors assume a growing importance in some groups. Global statistics demonstrate that 1%-3% of the world population have communication problems due to deafness. Hearing aids for children should be equipped with an automatic intensity control to prevent additional acoustic damage; otherwise children will refuse to wear the aid. In the aged, recruitment and sensitivity in the high frequencies create problems. Slow adaptation and schooling, at least for five weeks, are advisable. In Denmark, for instance, schooling is compulsory for all who apply for a free hearing aid from the state.

In order to differentiate lesions of the auditory nerve from those of the organ of Corti, Hofer [135] worked out a bone-conduction test by means of the tuning fork. A 128 cps tuning fork is applied to the mastoid bone on the deaf side, and the Bárány drum is used for masking. There are three possible responses: 1. When the tuning fork is applied to the mastoid of the deaf side, the tone will be localized in the good ear. This is indicative of lesions of the auditory nerve. 2. Without masking, the tone is heard in the deaf ear; as soon as masking is applied, the tone is no longer heard. 3. Even with masking the tone is localized in the deaf ear. The second and third types of response occur with damage of the organ of Corti. In such cases of perceptive impairment the tuning fork tone is heard in the deaf ear by bone conduction. This test is of aid in the differential diagnosis of perceptive deafness. It is an interesting finding that in a case of bilateral clinical deafness the subject was able to perceive on one side the tone of the fork by bone conduction; he did not only distinguish vibration and tone, but could also reproduce the pitch correctly.

The loudness recruitment phenomenon is only one element in a complex derangement of cochlear function due to end-organ disease, reports Hood.[139] The other important factor is the "relapse" phenomenon which occurs in response to tones of long duration. This means that in end-organ disease, the initial response to tones of short duration may approximate or even exceed the normal if the stimulus level is high; but this response is not sustained if the stimulus is continued and it may show an abnormal tendency to relapse. With sustained stimulation one may have elimination or even reversal of recruitment.

Modern aspects of the recruitment phenomenon are reviewed by Lansberg.[167] Differential diagnosis in cases of perceptive deafness can usually be based on the absence or presence of recruitment. In the prescription of hearing aids a thorough knowledge of what recruitment does to speech intelligibility is of paramount importance. Fatigue, adaptation, and trauma have their repercussions on hearing. Recruitment testing is one of the means to reveal the intricacies of these temporary or permanent hearing impairments. The middle-ear muscles can change the impedance of the sound-conduction apparatus. The change varies for the different frequencies and for bone and air conduction and depends on the status in the cavum tympani. Partial recruitment in bone conduction and in conduction deafness are discussed. Newer testing techniques are presented. Hood's findings of accelerated prestimulatory fatigue in cases of Ménière's disease were not confirmed by tests in cases of acoustic trauma. A diagrammatic representation of cochlear relations in both normal and pathological circumstances is tentatively advanced.

The accurate study and knowledge of the cochlear reserve is of great importance, although there is no current audiometric method enabling an accurate evaluation. It is usually evaluated by measuring bone conduction, especially for sound waves with frequencies of 1,000 and 2,000. This shows the functional disturbance, however, is marked by the presence of an irreversible fixed lesion, on the one hand, and a merely functional disorder which may readily disappear, on the other. Methods enabling a more accurate identification of the irreversible damage by variations in bone conduction were devised by Mayoux and Martin.[189] Fenestration and massage of the Eustachian tube resulted in an unanticipated improvement in bone conduction. The merely functional and reversible impairment of bone conduction may be demonstrated by intravenous injections of distilled water and hypertonic saline solutions as well as by injections of cocaine into the tympanum. There should also be other methods, as effective as sympathetic block and the use of certain ganglionic blocking agents.

The use of white noise is suggested by Palva [212] for general use in masking because of its efficiency and predictability. The masking characteristics of nonthermal type

of noise, supplied in commercial audiometers, are shown to be inefficient relative to the over-all sound-pressure levels. The interaction between the test tone and the masking note may result in a large dispersion of the masked thresholds. The attenuation across the head for air-conducted sounds in monaurally deaf persons is shown to be 50-55 db. for an average size receiver covering both ears, and about 60 db. if the opposite ear is kept open: the tones are transmitted via bone conduction to the opposite ear. Masking should be employed in all bone-conduction tests, since in bone-conduction tests the head vibrates as a whole. About 80 db. white noise can be applied to the opposite ear without reducing the sensitivity of the ear under test because of cross masking.

The well-known importance of convenient masking is emphasized by Kietz.[157] It is impossible to stimulate one ear separately. The diminution of the sound pressure from the stimulated ear to the other is of the order of 15 db. by bone conduction, and of 45 db. by stimulation with ordinary earphones (principally air conduction). The undesired stimulation of the nonexamined ear is called "acoustic by-pass." The masking effect of a noise depends on its composition and the frequency to be masked. Using white noise, the threshold elevation of the masked tone is directly proportional to the noise level, starting at the threshold of audibility. This is valuable for 1,000 cycles. Other frequencies are masked only by a noise level of 5 to 30 db. above threshold. Therefore white noise is not the ideal masking noise. It would be easy to construct a special kind of noise having the same masking effect on all frequencies for normal ears. In pathological cases all conditions are completely different. That is why the system of a general effective masking noise is not recommended. The difficulties may be overcome by a narrow-band masking noise centered on the test frequency and variable with it. Such a noise is called fully effective if its masking effect starts at its threshold. In measuring the bone conduction, increasing gradually the masking noise on the better ear raises the threshold of the worse ear as long as the hearing is done in reality by the nonexamined better ear. A very loud masking noise may reach the examined ear by the acoustic by-pass and raise again the measured threshold. The real threshold is therefore difficult to get by the method of gradually increasing the masking noise. In all cases of asymmetrical hearing the usually registered bone-conduction curves must be evaluated critically. A new system of regulating the intensity of the narrow-band masking noise is suggested: the lowest noise intensity to mask the acoustic by-pass on bone conduction is first measured in every case. Then a mechanical connection is fixed between the two controls of the tone-and-masking intensity. The difference of these values is therefore constant, sufficient to eliminate the acoustic by-pass without creating any undesired cross-over masking of the tested ear.

The old problem of masking is reviewed by Langenbeck.[165] Physically correct writing of the audiogram in absolute threshold values instead of the generally used hearing-loss curves simplifies the masking problems and gives the possibility of comparing the calibrated masking-noise intensity to the intensity of the test sound. Over-hearing by bone conduction from one ear to the other attenuates the sound energy by 10 to 20 db. Bone conduction over-hearing of the lower frequencies is increased by covering the ear with the normal earphone. Over-hearing exists not only when the bone conduction receiver (vibrator) is used, but also during the use of the air conduction telephone. Over-hearing is then produced at intensities of 60 db. and more above threshold. It is probably caused by stimulation of the crossed ear by bone conduction. After a careful analysis of several cases, the author lays down the principal rules to be observed in masking. In perception deafness the intensity of the masking noises for bone conduction tests is the same as or slightly louder than the air conduction threshold value of the poorer ear. In conduction

deafness the conditions are more complicated. It is advisable to begin with testing without masking. The ear with the better bone conduction is then masked. The masking intensity depends essentially on the air-conduction curves of the better ear and the bone-conduction curves of the poorer ear (under test). Each case needs a careful individual analysis to avoid over-masking by stimulating the ear under test by a too intense masking of the nonexamined ear.

Reconstructive Surgery of the Middle Ear

In operations for congenital atresia of the auditory canal, Zöllner [306] recommends sparing the auditory ossicles as much as possible. A chain of auditory ossicles with good mobility gives a better chance of satisfactory hearing than removal of the ossicles before the tympanic cavity is closed with a flap. Hearing is often remarkably good following the formation of a simple antrum fistula and removal of the rudiments of the malleus and incus. This is because the sound path leads through the antrum, and the distance between the windows is an important factor in obtaining the phase effect of sound waves. In the usual radical mastoidectomy, sound waves strike both windows simultaneously. Fenestration is advisable only when the stapes is fixed by malformation or adhesions or when it is impossible to establish a connection between the flap and stapes. Despite the absence of the drum, the ossicles might still be mobile. The flap or graft should then be placed on the incus. This has the advantage that the graft need not be placed as deeply into the cavity and that the body of the incus serves as a good nutritional support. In the majority of cases, however, the malformed chain is firmly fixed, particularly at the site of the fan-shaped ligament. This is easily separated. Surrounding soft parts may be interposed to prevent reunion. Another point of fixation is between the malleus and adjacent bony mass. Here good mobilization is possible by removing the bone until the malleus is completely freed. The mucosa of the middle ear and ligaments have to be carefully preserved to avoid adhesions and to insure nutrition of the ossicles. As to the deformed external ear, the canal must be wide and permanently open. The rudimentary ear is situated too far in front and must be repositioned posterosuperiorly so as to catch sound from ahead and to prevent rain and perspiration from entering the canal. Vertical incisions are made in front of and behind the rudimentary auricle. The auricle can be lifted like a bridge. A crescent-shaped incision, except for the middle third, is made behind the auricle, thus leaving a small pedicle connected to the scalp. After forming the bony external duct one buries this flap into the canal, lining its posterior wall. The auricle is pulled back and sutured. The buried flap is fixed to the bone with a silver nail since it tends to retract. A full-thickness graft is placed over the antrum and ossicles. Thiersch grafts complete the remaining raw surfaces.

The surgical correction of congenital anomalies of the external and middle ear is now an accepted procedure. Derlacki and Shambaugh [63] place emphasis on the embryologic development of the ear. Congenital anomalies of the sound conductive mechanism develop between the second and seventh months of embryonic life. The particular features of each individual case must be studied and the surgical technique fitted to the condition encountered. In most instances they prefer the atticotomy approach. Twenty-six ears were subjected to operation: nineteen had atresia; five a formed tympanic membrane, and in fourteen cases the drum was replaced by a bony plate. Hearing was maintained in twenty-one cases.

The surgical treatment of atresia auria congenita is reported by Ruedi [240] on a total of 19 ears—in 12 cases of bilateral ear atresia and 1 case of unilateral atresia. Nine of these cases were uncomplicated atresia; four were atresia combined with mandibulofacial dysostosis. In 10 out of 18 ears a good to adequate hearing gain was first

achieved by the Pattee method (creating a new meatus and removing the incus to mobilize the stapes). In 7 out of 10 Pattee cases the operatively achieved hearing gain was lost in the course of several years. By revision and fenestration, the initial hearing gain could be restored or exceeded in three cases. For uncomplicated cases of atresia the two-stage fenestration operation combined with stapes mobilization and possible resection of the stapes head in the first phase is recommended. Atresias combined with skull malformations should be fenestrated in a one-stage procedure.

Further contribution to the surgery of atresia auris congenita is reported by Rüedi and von Schulthess.[244] Report is made on 19 atretic ears operated on by a two-stage operation technique. If the plastic operation of the middle ear with extraction of the incus (Pattee-operation) does not improve hearing, fenestration is done at a second stage. In atresia combined with malformation of the skull a one-stage fenestration is preferable.

Theobald[272] states that any child with congenital atresia of the external auditory canal should have the benefit of modern temporal bone surgery. The chances of obtaining serviceable hearing in the ear operated on are very good, and, if that goal is not attained, a secondary fenestration operation may be done at a later date. The malformed malleus and incus should always be removed. If the stapes is fixed, fenestration surgery may be done later. Concern over the location of the facial nerve should not be a deterrant, since it is usually in its normal position. The period of postoperative care is shortened by skin grafting the new canal about 10 days after the original surgery. The canal should be made at least three times larger than it will ultimately be, for a great deal of scarring and contraction occur.

Holmes[136] reviews the embryology of this deformity. The operation of Pattee and fenestration are very useful procedures for the improvement of hearing, but a 30 db. hearing loss can be expected to remain. Therefore, surgery is advised only in bilateral hearing defects. Deformities of the auricle may present serious problems. Unfortunately prostheses have not proved successful due to changes in color with temperature variations or exposure to the sun.

Perforations of the drum were closed in 48 of 53 cases by Schrimpf[252] using amniotic membrane. The average hearing gain was 18 db. The technique is as follows: Under aseptic technique large pieces of combined amnion and chorion are placed in a sterile brown glass jar containing Ringer's solution with 1:1,000 merthiolate. The membranes are stretched out on a sterile towel. The chorion is separated from the amnion by stripping with a sterile forceps and scraped with a dull table knife. One must remember which side the chorion was adherent to, since this is the mesodermal surface which will be applied against the drum. The amnion is now cut into roughly circular pieces so as to fit into a Petri dish which has been previously fitted with a piece of corrugated cardboard thoroughly impregnated with paraffin and then autoclaved. Next a piece of damp membrane is stretched over the cardboard and held in place with sterile pins bent at a right angle to permit closing the lid over them in the Petri dish. One places the ectodermal (fetal) side of the amnion against the cardboard. The cardboard and the stretched membrane plus a small waxed paper container of calcium chloride are placed in the Petri dish and covered. This is allowed to dry 48 hours or longer. Then three leather workers punches Nos. 6, 7, and 8 ($\frac{5}{32}$, $\frac{3}{16}$ and $\frac{1}{4}$ in.) are used to make discs. One each of these sizes is placed, ectodermal side up, in the center of a square of sterile waxed paper, which is folded and stored in another Petri dish until needed. The ear canal is cleaned. Next a tampon of sterile cotton saturated with Grey's solution (10% cocaine in equal parts 95% alcohol and aniline oil) is placed against the drum for 10 minutes. Next a fine cotton-tipped applicator is dipped in 50% trichloracetic acid and then applied to

the edges of the perforation. A proper-sized disc of amnion is picked up with the applicator. This consists of a No. 24 steel wire 10 cm. long, the end of which has been fashioned by small pointed pliers into a loop of suitable size. This loop is then bent at right angles to the rest of the wire. The loop is flamed in an alcohol lamp, then dipped into sterile mineral oil. The oiled loop is then applied to the ectodermal surface of the selected amnion disc which at once adheres to the wire. Via a large speculum, the disc is guided and placed over the perforation.

The prosthetic treatment of a traumatic rupture of the eardrum during the Korean war was described by Sjöberg.[264] The greatest danger is secondary infection, leading to meningitis. Treatment with ear drops, local antibiotics, etc., augments the danger of infection to 75%. The best treatment is "Look, but don't touch." In special cases prosthesis can be used, but only if the ear is entirely dry. If the perforation exists more than four months, there is a danger that the epithelial cells may show signs of metaplasia, and prosthesis may be the cause of cholesteatoma formation. The prosthesis used in the Tokyo Army Hospital was copy paper with 96% alcohol. Paper prosthesis must remain for three weeks at least. The prosthesis serves only as a supporting material, a kind of bridge to the new epithelium.

Fournier and Rainville[93] published a consideration on the artificial drum. They used three types of artificial drum: (1) the tympanic prosthesis for the obturation of perforation of the drum; (2) the retrotympanic prosthesis; impregnated cotton introduced into the tympanic cavity and pushed in the niche of the round window; (3) ossicular prosthesis, that is to say, a nylon stalk intended to assume the function of the chain of ossicles. The results are sometimes very good, and with the first two types the authors obtained audiometric gain frequently of 30 or 35 db. In certain cases the effect of the artificial drum may be enhanced by the additional use of electronic hearing aids.

On the basis of personal experience with tympanic prosthesis the functional and clinical usefulness of these prosthesis is confirmed and the optimum indications enumerated by Clerici and Manzini.[51] Non-secreting mesotympanic perforations, caused by diseases of the middle ear and accompanied by transmission or mixed hypoacusia of not less than 25-30 db. deficiency over the complete tone scale and not more than 70 db. at conversation loudness, are considered the cases which may be expected to obtain the best results with the use of tympanic prostheses.

Chronic otitis media is treated by Biber[23] by "therapeutic filling." The ear is cleaned by syringing; polyps are removed and 10% silver nitrate is applied. A pledget of cotton soaked in an ointment composed of camphenol and camphonaphthol aa 3 gm., guaiacol 2.5 gm., iodoform 7.5 gm., and anhydrous lanolin 50 gm. is pressed against the perforation. It is changed every 48 hours, later every three to four days and even longer. When the ear is dry, residual epithelium at the perforation margin is removed with silver nitrate or curettes. Blood from the ear lobule is allowed to clot and is then transferred with a fine pipette to cover the perforation. The external canal is then plugged with cotton for five to six days. The dried clot is removed in two weeks. The perforation is usually closed.

Chronic suppurative otitis media without cholesteatoma is treated through the Eustachian tube with the aid of the catheterizing Eustachioscope by Pitman.[218] Suspensions or solutions (1.5 cc.) of freshly prepared 30% sulfathiazole sodium plus one of the antibiotics are used. The instrument consists of a nasopharyngoscope and two catheter guides (R or L) each of which can be mounted on the nasopharyngoscope with the aid of a sleeve. The catheter used is a 12 in. section of a No. 4 ureteral catheter having a forward outlet. The response to treatment, when effectual, was very rapid. The irrigation is done from within out and

is much more effective than instillation of medications into the external auditory canal.

The closure of dry central perforations of the drumhead is performed by Zöllner [305] by removing epithelium from the edge of the perforation, from the outer surface of the drum remnant, and from the adjacent external canal wall with care to save the connective tissue layer of the external canal and the annulus tympanicus. The latter is to ensure preserving the vascular supply. A portion of the promontory mucosa may be removed to provide another source of nutrition for the graft. If the tympanum is deep, small inserts of absorbable gelatin sponge (Gelfoam) soaked in penicillin are placed for supporting the graft. After placing the grafts, one holds them in place with absorbable gelatin sponge, and the sponge is then left in place eight days. In all large grafts the external canal is kept packed two to three weeks to provide warmth and moisture for the grafts. Before doing this surgery, one must determine if the ossicles are functionally intact, and progressive disease processes such as caries and cholesteatoma must be ruled out. The use of the acoustic probe is advised. If the ossicles are not functioning, the epitympanum must be opened, the pathological tissue removed, the defect diagnosed and, if possible, corrected. Once the epitympanum is opened, it is impossible to restore an air-filled epitympanum. If it is closed with a graft, the ossicles adhere to it. The epitympanum is left open and allowed to heal as rapidly as possible with minimum scar formation. The exposed head of the malleus vibrates in opposite phase to the drumhead but this effect is small, about 10 db. The tympanum itself must be closed to the external canal. Preservation of the ossicular chain even when partially destroyed is emphasized. If a cholesteatoma has not invaded the tympanum, tube, or labyrinth, the epitympanum is opened widely, the matrix is left in place and the process is permitted to heal by itself. Granulations of the ossicles are not touched. Impressive functional results were obtained with this conservative approach. If expected improvement in hearing does not occur postoperatively, one can determine the extent and location of the residual disease and then proceed to correct it with a minor procedure. If the tube is patent and the tympanum tightly closed, hearing impairment is then likely due to interruption or fixation of the ossicles. Removal of the adherent or defective incus and, if indicated, also the head of the malleus, followed by an establishment of a connection between the mobilized upper margin of the eardrum and the stapes should give a satisfactory result with a loss of 20-30 db. Bony adhesions in the annular ligament are rare, and therefore fenestration is seldom needed. In the rare cases where the cause of a constant middle-ear suppuration with a central perforation is a chronic mastoiditis and where the epitympanum is not markedly affected, a simple antrotomy without opening into the external canal should be done. Later a transmeatal plastic closure of the perforated drum is performed. If, owing to crowded conditions, the posterior wall of the external canal has to be removed in these cases, then the aditus may be closed with a graft while the mastoid is left open to the external canal.

Improvement of hearing after the atticoantrotomy is reported by Černý et al.[42] Atticotomy gave rise to hearing improvement in 54% (radical operation 42%); no changes were observed in 34% (radical operation 42%), and deterioration in 6% (radical operation 15%). The younger the patient the better are the prospects of improvement of hearing.

Factors influencing the hearing result in surgery of chronic otitis media are discussed by Thorburn.[273] Early surgical intervention is justified in properly selected cases, especially of cholesteatoma, with a view to conserving hearing. This applies even where the ear in question is the only ear with useful hearing. Especially in cases of cholesteatoma, a local tissue reaction can occur within the tympanum, limiting the spread of the disease. If this is respected, it can later be converted into a healed mobile

false tympanic membrane. One must avoid leaving any actual or potential blind pockets which may subsequently fill with disease. The ideal is to create a mobile actual or false tympanic membrane in functional continuity with the Eustachian tube and covering one or both labyrinth windows. If there is no incus or malleus, an intact stapes is an asset, especially if the mobile false membrane covers and is adherent to it.

In surgery of the mastoid for a chronic inflammatory process, one should aim for a technique that will conserve or improve hearing. According to Baron,[19] this technique should be designed to produce a mobile stapes and an unscarred round window in a sealed tympanic air space in continuity with the Eustachian tube. The incidence of cholesteatoma is high, and it is important to recognize that the matrix of the cholesteatoma can be converted to a useful structure, for the matrix has sealed off the tympanic space in an ear with good hearing. Leaving this matrix undisturbed will conserve or improve the hearing.

According to Bekker,[22] the mechanics of sound transmission in the middle ear have been understood for a long time, and great importance was attributed to an intact ossicular chain. The fenestration operation has proved that only a certain amount of decibel loss may be ascribed to the ossicular chain. Beyond a 30 db. loss, pathologic changes affecting the windows (fibrosis, obliteration) must be suspected. The author strongly advocates early operation for chronic otitis media, in order to prevent increasing damage to drum and ossicular chain. When drum and ossicles cannot be salvaged, every vestige of diseased tissue should be removed from the middle ear. When marked deafness develops after the operation, fenestration may improve hearing remarkably, provided bone conduction has remained good.

Attic disease and its treatment is discussed by Dingley and Zweifach.[68] This condition is usually found in the acellular type of mastoid, where in its acute stage it can be so silent in comparison with that in the cellular mastoid that it is often overlooked. It is often bilateral. Two types are described: the classical attic cholesteatoma and a second benign attic disease with granulations in attic and aditus, intact ossicular chain, and in which ankylosis between malleus and incus may occur. Attic cholesteatoma is prone to suffer sudden exacerbations of activity during which the hearing may deteriorate suddenly and sharply, but more important is the fact that such activity is frequently the herald of an approaching extension of infection to one or other of the danger areas near by. Whether hearing in the affected ear is good or bad, surgery is the safest method of treatment. The modified radical mastoidectomy is ideally suited for treatment of attic cholesteatoma since it renders the ear safe, completely eradicates the disease and preserves the hearing at the highest level possible. Results of this surgery in 23 cases of attic disease and in 15 cases of posterosuperior marginal disease are described. Hearing improvement was obtained in 17 cases, no change in 18, and deterioration in 3 cases.

Epitympanic chronic otitis media should be operated upon as early as possible provided that it is still limited to the epitympanum and the rest of the tympanic cavity is sealed off by a connective tissue membrane reports Müller.[203] In early cases of secondary cholesteatoma with posterior marginal perforation a conservative radical mastoidectomy is the procedure of choice. In the aetiologically and clinically different primary cholesteatoma with Shrapnell perforation, another operation is advanced: the attic is opened from the processus zygomaticus backwards to the antrum, up to the posterior margin of the cholesteatoma so that the cholesteatoma can be extirpated as a whole and all the granulation tissue excised. The auditory ossicles must be respected. The bone wound is covered with a skin flap. In this way the ear will heal quickly and the hearing very often will improve after the operation.

Conservative radical mastoidectomy to preserve hearing is recommended by Falbe-

Hansen.[76] The endaural incision was used. The attic was cleared of granulations. The tympanomeatal plastic flap was placed, as in a fenestration operation. If the ossicles were fixed by adhesions, buried in granulations, or were carious, the incus and head of the malleus were removed (17 cases) and the middle ear cleared of granulations and fibrous tissue, especially around the fenestra and Eustachian tube opening. Drum perforations were closed with thin Thiersch grafts slightly larger than the perforation and placed on a thin film of fibrin. Of 19 cases reported, 7 obtained hearing improvement; in 9 the hearing remained unchanged, and in 3 it deteriorated. In 15 dry cavities were obtained. In cases where hearing improvement is of decisive importance (marked hardness of hearing in the other ear) and the stapes fixed, a fenestration operation may be carried out when the cavity has become dry.

Goto[117] proposes a new method in the technique of mastoidectomy with which he has been successful not only in preservation of hearing but in bringing about an improvement. He calls this method "enhanced hearing modified radical mastoidectomy." Indications for operation, technique, and results of operations are reported. He also describes a method of skin grafting for early epithelization of the operative wound which has been simplified and made practical. As the grafting is accomplished at one sitting, the time required for operative procedure is considerably shortened.

Exposition of the different forms of chronic otitis media and of the modern surgical treatment (under microscopic magnification) of this disease is discussed by Kecht.[154] Conservative radical mastoidectomy and tympanoplasty give good chances for conservation or improvement of the preoperative hearing along with final removal of the inflammatory processes. It is advisable to operate early and not to wait until serious changes in the middle ear have been established.

Preservation of hearing in surgery for chronic ear disease is emphasized by Juers.[152] In properly selected cases a modified radical mastoidectomy preserves serviceable hearing and at the same time eliminates attic pathological changes. The following prerequisites are essential for a good hearing level after surgery: an air-containing inflatable space over the round window, an intact pars tensa, no fistula from the tympanic cavity into the attic, an intact, or unimpeded ossicular chain, or, if the incus is removed, contact between the pars tensa and the stapes should be assured. If such contact exists before surgery, it should not be disturbed. If such contact is not present it should be established by placing a tympanomeatal flap onto the head of the stapes (myringostapediopexy). If there is considerable conduction deafness before surgery, the incus and malleus head should be removed and myringostapediopexy accomplished. If serviceable hearing is not restored after complete healing takes place, a secondary fenestration might be considered at a later date if the need for serviceable hearing in the ear in question is sufficient. Each case is an individual problem, and the surgical management is dictated by a thorough study of the hearing before surgery and careful observation of the pathology and status of the ossicular chain during surgery.

The effect of various operations on hearing in chronically discharging ears was reviewed by Palva and Siirala.[214] They suggest that cases of subacute mastoiditis should be subjected to a simple mastoid operation after a period of conservative treatment not exceeding three weeks, to avoid permanent loss of function due to adhesive otitis or toxic degeneration of the inner ear. Discharging ears with a perforation in Shrapnell's membrane, or in the lateral attic wall only, should be subjected to modified radical mastoidectomy if the preoperative hearing loss does not exceed 40 db. When the hearing level is 20 db. or less, the ossicular chain should be kept intact; in losses of 20-40 db. the incus and head of the malleus can be removed without lessening the chances of serviceable hear-

ing. Radical mastoidectomy should be limited to cases of more than 40 db. preoperative hearing loss. In a considerable number of these cases, a practically useful level of hearing can be obtained and there is no significant hearing loss after the operation. In cases of chronic middle-ear suppuration with good cochlear function there are still chances of improving the hearing through operative measures, in spite of possible fixation or interruption of the ossicular chain. When the stapes is mobile and the round window is not occluded, an attempt should be made to reestablish the sound-conduction mechanism. If the stapes is fixed, fenestration should be considered. The matrix of the cholesteatoma should be removed in all cases in which the incus and the head of the malleus are removed.

If the primary hearing loss in the most important frequency range is less than 20 db., conservative operation should, as far as possible, be performed to preserve the preoperative level of hearing, report Palva and Ojala.[213] If the primary hearing loss is 20-30 db., a modified radical mastoidectomy with removal of the incus and head of the malleus can be made without risk to the hearing. If the preoperative hearing loss exceeds 30 db., conservative operation is no longer indicated as a rule. The modified radical operation aims at correction of hearing by reconstructing the tympanum. This can be done in various ways: If the pars tensa is unperforated, it can be pressed directly against the stapes; a perforation in the pars tensa can be covered with a skin flap, or a separate skin graft can be applied to the site of the tympanic membrane.

A case of recurrent chronic suppurative otitis media is recorded by Pruvost[224] in which the hearing was greatly improved by removing the head of the malleus and leaving the incus in position. There was good bone conduction. Suppuration was limited to the attic region. The hearing improvement is attributed to the lowering of the inertia of the ossicular chain.

A discussion is presented by Zanotti[302] of auditory function in patients subjected to a conservative-radical operation in order to determine whether, during the first two weeks after intervention, the changes of the physical conditions of the middle ear brought about by the operation lead to a deterioration of auditory acuity. In a previous study during the first period after fenestration, it was concluded that the changed capacity to perceive high tones depends, in case operative trauma is reduced to a minimum, not on postoperative aseptic labyrinthitis, but on the changed conditions of transmission of sound through the middle ear. On the basis of various functional elements, it is concluded that in patients subjected to conservative operation on the middle ear, there is, during a longer or shorter period after operation, a changed capacity of hearing high-frequency sounds, the same as in fenestration patients. This defect can only be attributed to the changed conditions in the middle ear.

A description is given by Vidau and Bormioli[281] of a method by which more rapid epithelization may be obtained after antroatticotomy. A pedicled dermoepidermal flap is detatched from the hairless region between the retroauricular groove and the hairline; the flap is given the shape of an inverted V. This flap is used to cover the antrum, the entrance, and the medial wall of the cavity; plasty of the duct is carried out, and the skin is sutured in the normal way. When after a month, the flap has definitely taken, the pedicle is cut and the small opening remaining near the apex of the mastoid is sutured. For bandaging, the authors recommend the use of iodoform gauze soaked in Gomenol oil, because of the antibacterial and antienzymatic properties of this oil.

Free skin grafts in aural surgery are again used, especially since antibioticotherapy, and also because the new tendencies of the ear surgery are toward a conservative functional surgery. The results which are obtained are generally good, report Ombredanne, and Poncet.[211] Their use is analyzed

in several types of interventions: (*a*) in the external canal stenosis, which relapse very often without skin grafts; (*b*) in the radical mastoidectomy, the postoperative period of which is simpler, and the hearing result is better; (*c*) in the functional ear surgery of tympanoplasty; (*d*) in ear atresia, in which the use of a skin graft is absolutely necessary. Ombredanne, in this later case, makes a "finger of glove" with a little sack of skin graft and places it in the operative cavity. This graft prevents stenosis and permits the passage of auditive vibrations toward the labyrinth.

In endaural radical operations an accurate covering of the bony walls with full-thickness skin should be attempted. The skin is taken from the retroauricular region and is carefully sutured on three sides to the skin of the external meatus. Meyer [195] reports that results in 19 cases are satisfactory.

Secondary skin grafting in fenestration and mastoid cavities is employed by Guilford and Wright.[118] On the 10th-18th day a split-thickness skin graft is removed from the inner aspect of the upper arm under local anesthesia. The grafts are applied through the meatus in six or eight strips in intimate contact with the granular area of the mastoid cavity. Packing is not necessary.

Mayer's [187] incision is a modification of the endaural incision for fenestration operation by prolonging it downward through the incisura intertragica to the base of the lobule. This gives a good view for this operation. A skin graft to line the mastoid cavity is taken from the upper leg. This incision is preferred because cicatrization is better than after retroauricular incision.

Acoustic Trauma

As a result of decisions by the New York State Court of Appeals [308] a number of claims were filed with the New York State Workmen's Compensation Board. Consequently the need for certain standards was felt and a series of questions was referred to a committee of technical experts. From the answers of the Committee one might note the following: Noises differ in "composition" as well as in intensity. High-pitched sounds are in general more injurious than low-pitched sounds of the same intensity. Most persons subjected to noises above 120 db. for several hours daily experience permanent damage to hearing after a number of months; noises in the 90-100 db. range cause permanent damage only to a few very susceptible persons after several years. The effect of distance on sound intensity should be measured in individual industrial situations. The only practical method is a sound-level meter, with octave band analysis of the several components of the noise. An appraisal of the permanence of an occupational hearing loss should be made after an interval of not less than six months after cessation of exposure to the injurious noise. Compensation awards are to be based on loss of ability to understand speech. Preemployment examinations, using a correctly calibrated pure-tone audiometer in a suitable room, are recommended and should be repeated after one week, one month, and three months of exposure, thereafter at yearly intervals.

The research activities of the subcommittee on Noise in Industry of the American Academy of Ophthalmology and Otolaryngology are beginning to produce valuable information in the field of acoustic auditory trauma, reports Wheeler.[294] Here are presented audiometric studies on factory workers before employment, which indicate that approximately 75% have normal hearing; 10% have a slight loss; 5% have a severe loss; 5% have asymmetrical loss of a slight degree, and 5% have asymmetrical loss of a greater degree.

Industrial noise and occupational deafness is weighed by Nelson.[207] The employer should use all possible diligence in surveying conditions in his plant, ascertaining whether detrimental noise is present and doing whatever possible to eliminate as much noise as possible. The first line of defense calls for the use of ear defenders; labor will need

to cooperate in making use of all safety devices provided. Physical examinations for loss of hearing should be made promptly and at intervals. If some employees are susceptible to noise, they should either be protected or transferred to less noisy work. When claim is made an employee should be able to establish what loss of hearing was present at a given time, and employers should be able to establish cause of loss and extent as of the beginning of employment.

Control of noise is a big problem in industry, reports Von Gierke.[283] There are three possibilities of control: (1) complete elimination of noise at the source; (2) attenuation of sound close to the source; (3) attenuating device placed in or over the ear. Unfortunately we have to rely in most cases on the third possibility. The dangerous noise level depends on three factors: (1) noise intensity; (2) frequency characteristics of the noise, and (3) duration of exposure. Only for a short time can 140 db. be tolerated without noticeable permanent damage; the 150-160 db. level is dangerous if suddenly applied. Noise levels in industry are dangerous only upon prolonged exposure. All ear protectors have shortcomings. Their maximum attenuating effect is 30-35 db. The next logical protector is a helmet covering the entire head; it eliminates about 10 db. in addition to the protection received from ear plugs; if the entire chest is covered, an additional 10 db. of sound transmission is excluded. Tightly fitting ear plugs without leak are the best protection, within average limits. Ear muffs which are comfortable do not provide protection up to the limit established by bone-conducted sound. Up to the 130 db. level protection may be obtained by what the author calls "typical plugs," "optimal plugs," and "plug plus muff," depending upon specific conditions. Above the 130 db. level there is no satisfactory solution for the problem of protecting continuously exposed personnel, and an attempt must be made to reduce the noise at the source.

Sataloff [250] contributes to the clarification of a common problem in routine industrial testing, the question whether auditory fatigue results in inaccurate hearing thresholds, impairs the validity of the test, or confuses its interpretation for both legal and research purposes. Thirty-two subjects were selected at random, all working in an area where the greatest noise exposure was 110-114 db. Readings were taken in the morning before the men reported for work, i. e., after approximately 16 hours of respite from intense noise; only 16 of the 64 ears tested showed normal hearing acuity, i. e., less than 15 db. loss in the 4,000 and 8,000 frequencies. Audiometric readings were repeated three times during the day: after three hours' exposure to continuous jet engine noise; after one hour of lunch time, and finally in the afternoon after three hours' exposure to noise. Audiometry was performed within 10-15 minutes after leaving the noisy areas. None of the subjects showed any significant auditory fatigue within the mentioned frequencies. It is concluded that in this specific industrial situation the factor of temporary hearing loss may be discounted if audiograms are done 15 minutes after routine daily exposure. The relationship between temporary and permanent hearing loss is not yet clearly understood. Each industry concerned with the problem of noise should conduct pilot experiments to determine whether auditory fatigue is a factor in routine testing, and to plan regular audiometric tests accordingly.

According to Majer,[179] no successful treatment exists for inner-ear changes due to noise and resulting in degrees of deafness. Therefore the selection of workers for noisy industries is of decisive importance. Before starting employment the employee should have an audiometric examination. Any existing impairment of the inner ear is likely to be aggravated, and subjects with otosclerosis or a familial tendency to it should be rejected. The same applies to workers with chronic otitis media and thickening of the mucosa, and polyps. Sensitivity to noise is determined by exposing the ear to a strong, precisely cali-

brated sound, measuring auditory acuity by means of the audiometer a short time thereafter. Protection of the worker against industrial noise is complicated because the communication with other workmen, the hearing of warning signals, etc., must not be made impossible. Therefore instruments have been invented which exclude only particularly dangerous frequencies.

The role of the otologist in the industrial hearing conservation program is discussed by Fox.[97] Calibrated speech audiometry is advisable for assessing hearing loss and determining degree of hearing disability. Hearing loss caused by prolonged exposure to loud noise may be considered as permanent six months after complete removal of the person affected from the area of loud noise. The objectives of a hearing conservation program are to conserve the hearing of workers, to avoid unnecessary economic loss to the employer, and to collect facts and data which will enlarge our knowledge of the defects of industrial noise and how they can be overcome.

The influence of electric and acoustic trauma in telephone service is discussed by Neuberger.[209] Differential diagnosis between these two factors is possible, since sound waves, when too intense, produce injury of the internal ear (hair cells in the organ of Corti). The resulting hearing trouble has the character of inner-ear deafness with complete recruitment. On the other hand, the electric shock transmitted by the earphones is an exceptionally observed phenomenon. The only case presented by the author shows a similar deafness of the inner-ear type. Recruitment, however, is absent. This is explained by the fact that the electric current produces pathological changes not in the organ of Corti, but in the cells of the cochlear ganglion.

Prophylaxis of acoustic trauma is an extremely perplexing problem. Simple ear plugs are impractical, as they impair hearing for conversational voice. Barton [20] tested the effectiveness of a new device, the "Lee Sonic Ear-Valve" which admits conversational tones but bars damaging noises. The device consists essentially of an ear plug which fits snugly into the ear canal; the stem contains a valve which moves with sound pressure. Upon loud and harmful pressure of sound the valve closes completely. The device was tested during target practice on a pistol firing range. Among a group of 34 subjects between 18 and 21 years of age, 18 wore the ear plug, and 16 did not. All ears were audiometrically tested before and after gunfire for 60 seconds. None of the subjects presented any visual evidence of trauma to the ear. Those who did not wear the device showed a characteristic loss in the high-tone frequencies due to acoustic trauma, while there was no loss in the high-tone range among those protected by the device. In addition, these subjects reported pronounced relief from the usual painful noise of pistol shots.

Pure-tone trauma and noise trauma are different forms of acoustic trauma. Rüedi [241] makes this assertion on the basis of experimentally produced histological changes in the animal ear and temporal deafness in the human ear. In the first stage of noise trauma, as in pure-tone trauma, the transition of physiological fatigue to pathological fatigue or stimulation deafness occurs abruptly above an individually varying critical intensity for white noise. The dip between 2,000 and 8,000 cps, following exposure to intense white noise, varies individually as far as the slopes of the curve and the localization of the maximum hearing loss are concerned. Noise-stimulation deafness begins constantly with a narrowing of the difference limen. As soon as a critical intensity of stimulating noise is surpassed, a pathological delay in the threshold recovery of the test tone 4,000 cps occurs. The critical intensity in the same subject fluctuates 5 db. plus or minus. There is no critical duration of a stimulating tone below its critical intensity. Threshold recovery for a test tone 4,000 cps after exposure to a white noise at different intensity levels near the critical intensity showed that recovery occurs within 6.5 minutes for intensities

up to the critical level and is delayed above this level according to the individual noise sensitivity.

Different types and importances of the acoustic trauma after exposure of the human and animal ear to pure tones and white noise are reported by Rüedi.[243] The inner ear of the guinea pig is much more vulnerable to intense sound than is the human ear. The audiometric and histological findings are different after pure-tone and after complex noise exposure. The former produces a hearing loss extending over 2 to 3 octaves above the traumatizing frequency, with its maximum at about ½ to 1 octave higher, sometimes associated with a displacement of the pitch (diplacusis). The hearing loss after white noise exposure starts at 500 to 1,000 cps, with its maximum between 4,000 to 8,000 cps. The different types of hearing loss after white noise exposure are examined by means of the Békésy audiometer. At 100 db. noise intensity the consecutive hearing loss embraces a large band of frequencies from 2,000 to 8,000 cps. The prestimulatory and the poststimulatory fatigue are registered at different intensities of the noise. There are two distinctly different reaction types: 1. The physiological fatigue is characterized by rise of the 4,000 cps threshold of short duration (6 minutes after 2 minutes of noise) without modification of the difference limen. 2. The pathological fatigue after more intense noise stimulation is characterized by (*a*) a narrowing of the difference limen at 4,000 cps; (*b*) a prolongation of the recovery time for 4,000 cps (14 minutes after 2 minutes of noise); (*c*) a very transitory lowering of the threshold at 1,000 cps with an enlarging of the difference limen. Immediately after the end of the noise stimulation, the 1,000 cps tone is heard better than before. The borderline between physiological and pathological fatigue is very sharply distinct, depending individually on the intensity of the noise stimulation. There is a critical noise intensity situated between 78 and 95 db. in the group of 10 persons tested by the author. This critical intensity which separates the traumatizing from the harmless noise is individually constant without any significant variation between the different tests on the same day. If the noise is even slightly below the critical intensity, the duration of noise exposure is of no importance for the symptoms of acoustic fatigue; there is never a pathological fatigue under these conditions. White noise above the critical intensity produces injury to the outer hair cells which are responsible for the perception of the weakest sounds. This test may provide a new means of estimating the individual sensibility to acoustic trauma.

Occupational deafness of flight radio operators is discussed by Rezende.[227] An analysis is made of the findings in two groups of persons: (1) those with more and (2) those with less than five years of service and 5,000 hours of flying. The findings are compared with those in a group of commercial pilots. It is concluded that aviation noise causes hearing loss and that this was present in a large percentage of radio operators. In the latter the contributory factors were aircraft noise plus radio noise. The percentage showing hearing loss was larger than that of commercial pilots of a similar group. The hearing loss was also greater than in radio operators employed on the ground over the same period, viz., five years. It is considered that radio noise causes occupational deafness which is accentuated by aircraft noise.

Until now the abnormal susceptibility of ears to acoustic damage was measured with tests of fatiguability. These are not very reliable because they do not distinguish between cellular (labyrinthine) and neural (retrolabyrinthine) fatigue. A new method is described by Lawrence and Blanchard[168] which measures the threshold of auditory nonlinearity by judging the level of beats between a loud fundamental tone and a second beat tone. It was found that ears susceptible to damage show a low threshold of nonlinearity. This was confirmed by animal experiments, using the method of cochlear microphonics before and after overstimulation with known sounds.

Lawrence and Blanchard [169] start with the hypothesis that the first sign of structural weakness of the ear is not loss of sensitivity, but something that appears at a much lower level of stimulation, viz., the failure of the ear to follow the vibratory pattern of the stimulating sound, or, in short, the introduction of acoustic distortion. The authors maintain, on the basis of experimentation with animals, that this tendency to distort sounds received is directly predictive of weakness of the auditory structures. They propose as a test of distortion the following: Expose the ear to a pure tone; superimpose upon this pure tone one that is 4 cps different from the second harmonic of the pure tone. If the receiving ear is distorting the original pure tone and introducing harmonics, the subject will hear a distinct beat, which is presumed to be the alternate, 4-per-second increment and decrement of the superimposed tone by the harmonic generated within the subject's ear. The level of stimulation of the ear is then lowered until the subject reports the disappearance of the beats. This point is presumed to be that intensity level below which the subject hears tones as they are patterned and does not introduce elements of distortion. The lower this point for a given subject, the more vulnerable he is presumed to be to loud noises.

The effect of noise on speech perception in clinical and experimental types of deafness was studied by Pestalozza and Lazzaroni.[216] "Experimental" types of deafness were obtained by means of a special apparatus which allows the examiner's voice to be reproduced with various frequency cuts. The experimental results obtained, and the comparison of these results with those obtained in various clinical cases of conductive and/or perceptive deafness, indicate that in perceptive deafness noise is not directly responsible for the decrease of the articulation score, but only for the threshold shift; in fact, even in the absence of noise the distortion, which is always present, produces a progressive worsening of perception as the intensity is increased.

The occupational hearing disturbances of the flying personnel is of complex nature. It starts with a pure cochlear lesion; secondary factors always complicate the clinical picture and may be improved by medical treatment. This paper by Robert [231] deals exclusively with the initial lesion, which touches as a rule the sounds between 3,000 and 6,000 cps, starting with the classical dip at 4,000. A typical and an atypical evolution are distinguished. In the typical cases, the 4,000-cycle dip is progressively enlarged, touching the very high sounds, but also the frequencies above a fixed point between 1,000 and 2,000 cps. The latter separates the normal and the impaired part of the audiogram. During this phase the hearing of speech is intact. The second phase which follows the first after a very variable interval is characterized by a moving of the "fixed point" against higher intensities or lower frequencies. The social hearing capacity may still be conserved. In atypical evolution, the lower sounds are rapidly touched with a consequent severe hearing loss in the conversational area.

Excessive noises and supersonic vibrations up to 160 db. and above are produced by modern aircraft and many affect the inner ear. This will depend upon the duration of exposure and individual susceptibility. It may cause reduction in hearing in special frequencies. Treatment and prophylaxis of barotraumatic damage is discussed by Langraf,[166] emphasizing the necessity of treating chronic Eustachian tube obstruction. Lymphatic tissue around the pharyngeal ostium should be destroyed by Crowe's radium treatment. For obstruction of the tube itself, x-ray treatment and intratubal bougies and radioactive isotopes are suggested. The need is emphasized for the use of obturators for all flying personnel exposed to excessive noise. Flyers with loss of hearing at some frequencies in pure-tone audiometry develop a kind of paracusis Willisi and have no trouble in receiving signals in a plane. This adds to the opinion that a pilot with a defect in pure-tone audiometry in aircraft noise is satisfactory.

Reger and Lierle,[226] using the Békésy audiometer, exposed human subjects to a 1,000 cycle frequency tone at 20 db. and 80 db. above threshold. The recorded temporary threshold shifts (TTS) were greater for the exposures at 20 db. than for 80 db. This was statistically analyzed and found to be correct for a significant number of persons. Mention is made of the possibility of the action of the intratympanic muscles, which may have been stimulated by the higher intensity, thereby protecting the inner ear, whereas at the lower intensity of 20 db. the intratympanic muscles may not have been activated. The results obtained possibly have a bearing on the physiology of these muscles as part of the protective mechanism of the human ear.

The destructive changes in the cochlea of guinea pigs induced by repeated explosive sounds (approximately 120 phons) were observed in two groups by Haruo.[128] The animals of the first group were either anesthetized or sleeping; those of the second group were conscious. The destructive changes were consistently greater in the first group. This is attributed to the loss of normal tonus and acoustic reflex contraction of M. tensor tympani and M. stapedius.

(To be concluded)

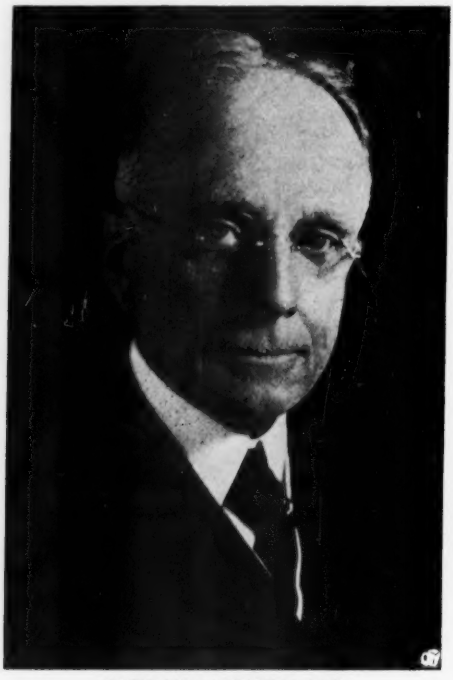
Obituaries

CHEVALIER JACKSON, M.D.

1865-1958

Chevalier Jackson, M.D., Sc.D., LL.D., L.H.D., died on Aug. 16, 1958, in his ninety-third year, after an illness of over a year. He had been a most helpful Associate Editor of the A. M. A. Archives of Otolaryngology from the time of its foundation until the beginning of his final illness. So passed one of the greatest, if not the very greatest, laryngologists of all time.

Space will not permit a detailed account of his life and works, his many accomplishments, literary and artistic productions, his inventions, his teaching career, and his service to humanity.



CHEVALIER JACKSON, M.D.

1865-1958

Beginning with the original work of Killian, he expanded the science of peroral bronchoesophagology into what it is today, in its many functions, from its original function of the extraction of foreign bodies from the air and food passages to its diagnostic values and to the care and cure of various pathological conditions. He invented and improved many of the original instruments used for these purposes, often building them in his own workshop. He set up a standard technique for the safe use of these procedures.

He was a master teacher, not only in the six institutions in which he held the rank of Professor in the United States, but in most of the countries of the western world; and many of the world leaders in this specialty became so because of his teaching, his lectures (often illustrated by his simultaneous ambidextrous blackboard drawings), and by his scientific articles in medical journals and his textbooks.

Dr. Jackson devoted the whole of a life of constant hard work, in spite of recurrent illnesses, to carrying on this great work for the relief of suffering and death. His hobbies were painting, in which he also excelled, and woodworking, and he was also very much interested in wildlife, and kept his property as a sanctuary.

Dr. Jackson was born in Pittsburgh Nov. 4, 1865, the son of William Stanford and Katherine Ann Morange Jackson, both born in Pennsylvania. In 1899 he married Alice Bennett White, and they had one son, Dr. Chevalier Lawrence Jackson, who, as all know, followed in his father's footsteps and is his successor at Temple University.

His medical education was at Jefferson Medical College in Philadelphia, from which he received his M.D. in 1886. One should read of his early struggles to forward this education in his Autobiography published in 1938.

Dr. Jackson became Professor of Laryngology in the University of Pittsburgh in 1912, in Jefferson Medical College in 1916, in the Graduate School of Medicine of the University of Pennsylvania in 1919, in the Medical School of the University of Pennsylvania in 1924, in Temple University School of Medicine in 1930, in the Woman's Medical College of Pennsylvania in 1934 (in 1935 he became President of that famous institution, the first of its kind in the world), and Visiting Professor in the Louisiana State University Medical Center the same year. In all but the first and last instances, new chairs of Bronchoscopy and Esophagoscopy were created for him. The terms of service in a number of these institutions were held simultaneously, but to all he gave devoted service. and in each one he established and equipped adequate departments where none had previously existed. In each one, also, he carefully trained his successor. All of these successors became famous laryngologists—i. e., Ellen J. Patterson at Pittsburgh, Louis H. Clerf at Jefferson, Gabriel Tucker at the Medical School and the Graduate School of Medicine of the University of Pennsylvania, Chevalier L. Jackson at Temple, and Emily Lois van Loon at the Woman's Medical College.

During his long lifetime of work, Dr. Jackson was the recipient of honorary degrees from seven institutions of learning, and held membership (or fellowship) in eighteen American scientific societies, and honorary membership in ten American and twenty foreign societies.

As an example of his great industry, he produced twelve textbooks, seven of which were translated into French, Italian, Spanish, and Japanese. He wrote six monographs, contributed chapters to thirteen systems of medicine and surgery, contributed 238 articles of his own, and 473 in collaboration with members of his various staffs, to medical journals over the world.

As an example of the appreciation his strenuous work received, he was the recipient, between 1924 and 1952, of fourteen U. S. and nine foreign medals and decorations. For the interest of the readers of this Memorial they are listed below:

1924—The Kiwanis Service Medal

1925—The American Medical Association Certificate of Honor for Exhibit of Bronchoscopic Work

1927—The Philadelphia (Bok) Award
Chevalier de la Légion d'Honneur (France)
Chevalier de l'Ordre de Léopold (Belgium)

1928—The De Roaldes Award in Laryngology of the American Larygological Association (first recipient)
The Henry Bigelow Medal, awarded by the Boston Surgical Society

1929—Officier de la Légion d'Honneur (promotion from Chevalier)
The Cresson Medal of the Franklin Institute

1932—The I. P. Strittmatter Award (for year 1927)
The Gold Medal of the Radiological Society of North America

1933—Commandatore del Ordine della Corona d'Italia

1935—Grão Mestre da Ordem Nacional do Sul (Southern Cross of Brazil)
The Medal of Honor, Academia Nacional de Medicina Rio de Janeiro

1936—Lauréat de l'Académie de Médecine (Prix Panneton) Paris

1937—Lauréat de l'Académie des Sciences (Prix Bellion) Paris

1939—Meritorious Service Medal of the State of Pennsylvania

1940—The Distinguished Service Medal of the American Medical Association

1943—The John Scott Award, City of Philadelphia, for Devising Instruments and Methods Useful in Bronchoscopy

1949—Medallion of Honor, Interstate Postgraduate Medical Association of North America

1952—Medallion, American College of Chest Physicians
The Russell H. Conwell Award, Temple University

Dr. Jackson was president of a number of scientific societies: in 1911, President of the American Laryngological, Rhinological and Otological Society, as well as Chairman of the Section on Laryngology, Otology and Rhinology of the American Medical Association; in 1917, President of the American Bronchoscopic Society, which he founded; in 1926, President of the American Laryngological Association; in 1934, of the Pan-American Medical Association; and in 1937, of the American Therapeutic Society.

In 1913 he was a Founder of the American College of Surgeons. In 1917 he served on the Medical Advisory Board of the State of Pennsylvania. He was Chairman of the Committee on Lye Legislation of the American Medical Association, which resulted in the passage of the Federal Caustic Poison Law, for which he had been working almost single-handedly for years, having seen so many of the horrible results of lye burns of the pharynx and esophagus. He had infinite patience and tenderness in caring for these tragic and difficult patients, many of whom were children.

For a number of years before his death Dr. Jackson had lived in semiretirement at the Old Sunrise Mill, his country estate near Schwenkville, some thirty-two miles from Philadelphia, coming to Philadelphia only for his lectures in his special courses in bronchoesophagology, which he had conducted annually for many years. He was by no means idle, however, and almost to the last was a a valuable reviewer of articles submitted to the A. M. A. Archives of Otolaryngology, also continuing to revise his textbooks and other scientific writing. Until recently he did much of this work floating in his home-made boat on the placid water of his millpond.

GEORGE M. COATES, M.D.

Abstracts from Current Literature

Ear

MYRINGOPLASTY: PLASTIC REPAIR OF EAR DRUM PERFORATIONS. L. PIETRANTONI, and E. BOCCA, Ann. Otol. Rhin. & Laryng. 66:1160 (Dec.) 1957.

In performing myringoplasty for perforations of central type without suppuration, the authors adopt the endaural incision with temporary displacement of the posterior canal wall skin. Advantages are better placement of a postauricular free-skin graft and ability to use larger grafts than would be possible with the transmeatal approach. A high degree of successful grafting is afforded by the meticulous removal of the cutaneous layer of the eardrum and part of the bony meatus, so that a large base of implantation for the graft is provided.

The criteria which indicate this type of intervention are stated by the authors as follows: dry perforations of the pars tensa of long duration; good bone conduction; improvement of air-conduction audiometry following application of a prosthesis over the perforation. In addition, it is occasionally proper to operate with benefit upon ears showing recurrent infection of the tympanic cavity or in those showing tinnitus which subsides on temporary closure of the perforation.

STEELE, Kansas City, Kan.

RESULTS WITH THE CHISELS IN STAPES MOBILIZATION. HAROLD F. SCHUKNECHT, A. BRUCE GRAHAM, and MARY ROSE COSTELLO, Laryngoscope 68:726 (April) 1958.

Schuknecht describes the methods he has used for mobilization of the stapes and analyzes the results obtained by each. By far the best results (72% of 72 cases) were obtained by the chisel method. This consists of the use of the Heermann chisel, placed directly on the otosclerotic lesion. An assistant then taps it with a small hammer. In the heavy otosclerotic lesions, the anterior crus may have to be sacrificed, but good results are still obtained from the columella effect of the posterior crus.

HITSCHLER, Philadelphia.

A GUIDE FOR STAPES SURGERY BASED ON A NEW SURGICAL CLASSIFICATION OF OTOSCLEROSIS. RICHARD J. BELLUCCI, Laryngoscope 68:741 (April) 1958.

The necessity of using the operating microscope in mobilization procedures is stressed. Only with this can the forces of mobilization be applied properly under direct vision to the otosclerotic area, i. e., the footplate. Other methods of mobilization, namely, transincudal and force applied to the head and neck of the stapes involve too great a chance of fracturing the delicate crura. Four months after the operation improvement continued in 55% of the cases where the force was applied to the foot plate, as compared with 29% where it was applied to the head and neck of the stapes.

Anatomical situations in which the surgical approach is difficult are discussed. A deeply recessed oval window with an overhanging facial nerve is one.

Palpation of the stapes may aid in determining where the otosclerotic lesion lies, so that the proper procedure may be used. Likewise success in mobilization can be determined by palpation plus visualization of the footplate and round-window notch.

Otosclerotic lesions are classified into four general types, depending upon the amount of sclerosis present and the ease of mobilization.

Specific methods to be applied to each are shown. Results are tabulated and discussed.

HITSCHLER, Philadelphia.

STAPEDIO-VESTIBULAR OSTEOTOMY IN OTOSCLEROSIS. MICHAEL PORTMANN and GILBERT CLAVERIE, Laryngoscope 68:797 (April) 1958.

Portmann and Claverie describe two rather unusual approaches to mobilization of the stapes.

A stapediovestibular osteotomy is performed. The incus is disarticulated from the stapes. The stapes is then removed from the oval window but not disconnected from its tendon. In order to prevent reankylosis, a graft consisting of a section of a vein is laid in the window, the stapes then replaced on the graft, and the incus upon the stapes. In very severe cases where the sclerotic bone completely closes the oval window, the crura of the stapes are cut,

leaving the head and crura attached to the tendon. This is removed. The oval window is then cleared of the sclerotic bone ("fenestrated"), the graft applied, and the stapes replaced, with the ends of the crura resting upon the graft.

Excellent results are claimed, although no case reports or statistics are given in this presentation.

HITSCHLER, Philadelphia.

ATTICOMASTOIDECTOMY IN CHRONIC OTITIS MEDIA WITH CENTRAL PERFORATION. ROBERT HENNER and COLETTE JEANTET, Laryngoscope 68:780 (April) 1958.

It is suggested that, with our modern methods of otologic surgery, the indications for operation on chronic supperative ears be revised.

One with a central perforation will respond to an atticomastoidectomy. This is really a complete single mastoidectomy plus a complete atticotomy with skeletonization of the labyrinth, dural plate, and ossicular chain. The posterior and superior canal walls are left intact. Granulations in the middle ear may be removed through the perforation. Hearing is usually improved by this procedure, and usually the perforation heals spontaneously. It is preferred over the older modified radical procedures where postoperative care was frequently a problem.

Case reports are given, together with a discussion of the pathology encountered.

HITSCHLER, Philadelphia.

PROFOUND DEAFNESS FROM NEOMYCIN SULFATE. ABRAHAM I. GOLDNER, New York J. Med. 58:2226 (July 1) 1958.

Goldner reports a case in detail to show how drugs used for various general bodily conditions may work irreversible harm to the ears (oculists have already made their observations as to the eyes). All of us in medical practice should recognize the sensitivity of both auditory and vestibular nerves to certain drugs, and govern ourselves accordingly. Cases are referred to in this essay which have been reported in the literature, testifying to the dangers inherent in administration of certain antibiotics and other drugs. This is no new subject, but needs especial emphasis at this time. Goldner's patient had a kidney condition and had been treated in hospitals with a variety of drugs. He had never had any difficulty with hearing (no family history of impairment) until he received neomycin sulfate. Sudden severe deafness ensued. But he was assured that this would be only temporary! It is now regarded as permanent after many examinations by otologists in and out of hospitals. "A hearing aid was ineffective." The static labyrinth was also involved.

ABSTRACTER'S COMMENT: It has been known for many years that quinine and the salicylates can impair hearing. In the early days of "606," there were many reports of deafness and blindness, probably due to impure arsenic. Terramycin, when it first was administered, took the blame for impairing the hearing. It was used in this case as well as neomycin. Were the two synergistic?

I. W. VOORHEES, New York.

ACOUSTIC AND VESTIBULAR DISTURBANCES FOLLOWING STREPTOMYCIN-TREATED TUBERCULOUS MENINGITIS IN CHILDREN. L. JAAKKO RANTA, Acta oto-laryng., Supplement 136, 1958.

This report is a study of 141 children who survived tuberculous meningitis. It concerns itself chiefly with 101 of these cases with 124 nonmeningitic controls. Purposes were to determine hearing loss after nonmeningeal tuberculosis, the incidence and severity of hearing loss varying with age in those affected by tuberculous meningitis, possible dependence of hearing loss upon the preparation, the dose, and the route of administration. The time of onset in terms of the stage of therapy, the incidence and severity of vestibular dysfunction, and finally the subsequent development of the disturbances observed.

Originally patients received 30-50 mg. of dihydrostreptomycin per kilogram per day I. M. Later the dose was 10-20 mg. Intrathecal strep was given concomitantly. Later added were para-aminosalicylic acid, isoniazid, and cortisone. Standard hearing tests and caloric tests were performed. Controls had an 18% hearing loss as compared with 62% in the regularly treated series. In the controls the hearing loss was slight. In the controls the dosage was low but the total dose very high over a long period. Total and subtotal deafness was present in 12% of treated cases of meningitis. As the daily dose of dihydrostreptomycin was reduced when adjuvant therapy came in, there was a corresponding decrease in the cases of hearing loss. However, at the same time the course of the meningitis also became more favorable.

Observations indicate that a daily dose of 10-20 mg. per kilogram of dihydrostreptomycin, with a total dose not exceeding 50 gm. seems fairly nontoxic.

Hearing loss was all perceptive. It was usually abrupt (57%). It was most commonly at the high ranges. Hearing loss was no higher in patients with papilledema. Most frequent time of onset was in the fourth month of therapy. Vestibular disturbances occurred in 52% of the meningitis cases. Generally there was both hearing loss and vestibular function loss. They did on occasion appear in one when the other was clinically normal. Changes were generally permanent. Further trouble occurred on occasion even after treatment was stopped.

It may be concluded that these unfortunate changes under the newer regimen and advances in therapy give hope for cure of tuberculous meningitis as a real possibility without any marked development of auditory or vestibular disturbances.

LEWY, Chicago.

ELECTRO-NYSTAGMOGRAPHY IN THE CALORIC AND ROTATORY TESTS. JAN STAHLE, Acta oto-laryng. Supplement 137, 1958.

The author describes a clinical study of electronystagmography in re caloric and rotational tests. The work covers 104 investigations in 91 instances of vestibular disease. He reviews the various features of nystagmus and the methods of testing. His work leads him to an interesting group of conclusions.

1. In Ménière's disease the direction of any positional nystagmus gives no indication of the site of the lesion.

2. In many cases diminished labyrinthine function is reflected in the number of beats, total amplitude, and maximum intensity, but not in the duration of the caloric reaction; further, clinical assessment should be based upon the difference in right-left sensitivity and between right-beating and left-beating nystagmus.

Caloric directional preponderance is often found in cases of peripheral vestibular disease, usually associated with diminished sensitivity, commonly reflected in both duration and intensity; the latent period may reflect D. P.

He finds postrotatory D. P. equally obvious; however, the clinical value of this is slight as it rarely provides information not available by full caloric testing. Even in cases that have a nystagmus already present in the test position, electronystagmography renders an assessment of labyrinthine function possible. An average value for the intensity of the present nystagmus is deduced, and from this level the effect of calorization is judged.

LEWY, Chicago.

Larynx

CONTROVERSIAL ASPECTS OF THE TREATMENT OF LARYNGEAL KERATOSIS. ROBERT E. PRIEST, Laryngoscope 68:766 (April) 1958.

The management of laryngeal keratosis is extremely difficult. In the first place the pathological interpretation of the lesion is apt to vary greatly as to its progression toward carcinoma, and its invasiveness.

Should x-ray therapy be used? Should it be used in nonmalignant lesions, with the hope of preventing malignant changes, the fact being kept in mind that future surgery, if necessary, is made more difficult?

The similarity of laryngeal and cervical lesions is discussed. It is concluded that there is no one treatment for all these lesions, but each must be assessed individually and treated accordingly.

That frequent inspections and biopsies are necessary, however, is agreed upon by all.

HITSCHLER, Philadelphia.

Nose

TRANSILLUMINATION OF THE NASAL SINUSES. F. H. MCGOVERN, J. A. M. A. 167:1620 (July 26) 1958.

The findings reported here are of the study of 100 cases with symptoms referable to the nasal sinuses by means of transillumination. There are many variables, such as the differences between men, on the one hand, and women and children on the other. In men, the heavier superior maxilla transilluminates poorly, while, in contrast, the x-ray may appear normal.

In women and children, the slighter bony structure of the same region may transilluminate clearly, while with x-ray the antrum may appear opaque. Transillumination is considered unreliable in women unless there is a definite difference between the two sides. A number of women and children in the series had what is called "masked sinusitis," where there were no clinical symptoms, but the findings of x-ray and antral irrigations were positive. The conclusion is reached that transillumination of the paranasal sinuses should be used only as confirmatory or supplementary.

SELTZER, Philadelphia.

RHINOPLASTIC APPROACH FOR DERMOID CYSTS OF THE NASAL DORSUM: REPORT OF THREE CASES. RALPH H. RIGGS, Laryngoscope 68:699 (April) 1958.

Less than 75 cases of midline dermoid cysts of the nasal dorsum have been reported. Various surgical procedures have been described to remove the lesions, all but one by external incisions.

By the use of rhinoplastic procedures in applicable cases there may be less visible postoperative defects. Each case of course has to be evaluated in itself and the procedure varied accordingly.

These cases are reported in detail.

HITSCHLER. Philadelphia.

THE FOREHEAD FLAP IN NASAL REPAIR. J. B. ESCOFFIER, Plast. & Reconst. Surg. 21:94 (Feb.) 1958.

The principal methods of nasal repair are reviewed, and the plain forehead flap chosen as being preferred by the greater number of operators, because of the simplicity of the procedure, the rapidity with which the operation can be accomplished, because of the fewer stages required; the safety of the method because of the ample blood supply to the flap, there being less chance of necrosis at the distal extremity. The flap is especially suitable since the texture and color of the skin is nearly the same; comfort to the patient is assured, as it is possible to see and to take food easily during the entire operation. Considering these advantages, objections which have been made do not seem to be significant.

The paper is amply illustrated by 16 figures, showing the operative steps, and two tables with details of the cases; 26 references to the literature are added.

SELTZER, Philadelphia.

LOSS OF COLUMELLA AFTER LEISHMANIASIS: RECONSTRUCTION WITH SUBCUTANEOUS TISSUE PEDICLE FLAP. A. DUARTE CARDOSA, Plast. & Reconst. Surg. 21:117 (Feb.) 1958.

Leishmaniasis is a relatively common disease in South America. It begins as a mucosal lesion in the inferior part of the nasal septum, near the columella; consequently this part of the nose is destroyed. Only the columella and septum may be affected, but this may cause retraction of the nose, with loss of the nasal tip, and even the alae and upper lip. The treatment must depend on the condition in the individual case. When there is loss of the columella combined with associated lesions, the operation chosen by the author is a median frontal flap which will correct all of the defects of the nose at once. The operation is described in much detail; four photographs are presented of loss of the columella and the tip of the nose; six photographs are shown of the left profile and full face, both before and after operation, others of the loss of columella and nasal retraction—more extreme, with added destruction of the alae, columella and upper lip—both before operation and after. Four before and after views of columellar loss are presented. Frontal skin flap with a subcutaneous pedicle containing the frontal artery is the method recommended, whenever a free composite graft cannot be used.

SELTZER, Philadelphia.

CORRECTION OF COMBINED ELONGATION OF NOSE AND RECESSED NASO-LABIAL ANGLE. J. A. CINELLI, Plast. & Reconst. Surg. 21:139 (Feb.) 1958.

This condition is spoken of as one of the most difficult rhinoplastic problems, for the correction of which many methods fail. The writer's own treatment is described in much detail and is illustrated by two cases, of which six photographs are presented, both before and after operation, in frontal, profile, and basal views; six drawings are also included of the successive operative steps and the final result.

SELTZER, Philadelphia.

TREATMENT OF DERMOID CYSTS OF THE NOSE. V. H. KAZANJIAN, Plast. & Reconst. Surg. 21:169 (March) 1958.

Six cases are reviewed which suggest that dermoid cysts seem to correspond with the various embryonic facial clefts. They occur about the nose, auricles, floor of the mouth, and the palate. There is little change of the contour of the nose, but a painless swelling is present over the nasal tip or near the glabella. The condition may not be evident at birth but becomes noticeable later, or sometimes earlier as a small pit over the dorsum, through which a fine probe can often be inserted, or fine hairs may be seen protruding from the pit, the location of which may vary in different instances. Diagnosis is made by the presence of protruding hairs, or by secretions which can be expressed. The size of the cavity can be determined by the use of iodized oil and x-ray. Cysts which do not have an outlet can be located by a considerable distortion of contour of the external nose, or by the presence of a soft swelling, which is painless unless infected. It should be differentiated from meningocele. Treatment is by surgery. The cavity is exposed and the cyst contents removed, care being taken to excise the membrane lining the cavity. Photographs and drawings illustrate the condition, these and drawings of a cyst, and photographs of the cyst contents. Five references to the literature are included.

SELTZER, Philadelphia.

Miscellaneous

ABRASIVE SURGERY IN NEGROES. R. D. MURRAY, Plast. & Reconst. Surg. 21:42 (Jan.) 1958.

Although the results of surgical abrasive treatment in whites may be somewhat variable because of pigmentary responses in some instances, the same treatment in Negroes is much more varied. The same sort of result occurs after skin injuries in Negroes, particularly following burns. Skin pigment over the area may be entirely lacking, or may be much increased. This is also true when split-thickness skin grafts are used. Experiments were carried out on the inner surface of the forearm, in which small pieces of skin were removed and photographs taken both before and after abrasion; then after abrasion another small piece of skin was removed to determine the depth of the abrasion. The epithelium was found to have been removed, but the skin appendages were still present. Pigment over the abraded area was absent for about a month; then the pigment began to return, first about the pores, then extending outward until the single pigment spots fused to become continuous over the entire surface, but irregularly, with lighter and darker portions. Histologically, the pigment granules in the cells were first fine, increasing in size until they occupied most of the cell. It is recommended that surgical abrasion be used on Negro skin always with reservations, particularly in "keloid formers," though if it is lightly done, the growth does not become keloid throughout. Negroes should always be told the possibilities following surgical abrasion and be allowed to make a choice. Photographs of both white and Negro examples are included, with eight photomicrographs. There are seven references to the literature.

SELTZER, Philadelphia.

A NEW LOOK AT THE ETIOLOGY OF CLEFT PALATE. E. J. STIEGLER and M. F. BARRY, Plast. & Reconst. Surg. 21:52 (Jan.) 1958.

This study is based on 164 families, mainly under the headings of Heredity; Incidence of Racial Groups; Associated Structural Deformities. A tendency to cleft palate was observed where there had been color intermarriage in the maternal antecedents. There may not be a specific gene for cleft palate, but instead, one for deformities in general, as club foot, spina bifida, supernumerary fingers and toes, malformed ears, atypical tongue, etc. Prematurity and multiple births were often associated with cleft palate; abortion and stillbirth may be followed by an offspring with cleft palate; poor nutrition was considered a "precipitating factor." Atypical conditions in the mother may be accompanied by the palate lesion. The Rh factor was not found to be related to the condition. There seems to be a chief factor of genic transmission—a common "malformation gene."

SELTZER, Philadelphia.

PLASTIC SURGERY IN THE TREATMENT OF RELAPSING LEISHMANIASIS OF THE FACE. Z. NEWMAN and I. KAPLAN, Plast. & Reconst. Surg. 21:112 (Feb.) 1958.

A condition is described here which is common in the Middle Eastern countries where leishmaniasis is endemic. Ulcers form, which become infected and may persist for 10 or 15

years, with the formation of scars and resulting deformities of the face. Treatment is excision of the ulcer down to the healthy-appearing underlying tissue, and covering the defect with a full-thickness graft taken from behind the left ear. In the authors' experience the graft takes, and the patient can be discharged on the 18th day. Two cases are illustrated by eight photographs taken both before and after operation. Two microphotographs of tissue sections are also included.

SELTZER, Philadelphia.

PALPEBRAL PTOSIS. E. F. MALBEC and A. R. BEAUX, Plast. & Reconst. Surg. 21:124 (Feb.) 1958.

Various methods for the correction of this condition are reviewed, but the authors prefer to use strips of fascia lata, because of their close application and the excellent results they have had with this procedure. Reasons for any objections were overcome by careful attention to suturing. The conclusions are based on 22 operations. Details are illustrated by the nine photographs of five cases, with before and after views. Some of the strips of fascia lata were autogenous, and some were from bovine source. Sixteen references to the literature are added.

SELTZER, Philadelphia.

JOHN BELASSA, THE GREAT HUNGARIAN SURGEON OF THE 19TH CENTURY. H. BERNDORFER, Plast. & Reconst. Surg. 21:143 (Feb.) 1958.

An interesting presentation of the work of an expert plastic surgeon in Hungary. Details are included of methods used by Belassa in plastic operations about the face and neck; also photographs and drawings showing his methods on the nose, mouth and eye in 12 figures.

SELTZER, Philadelphia.

THE DEVELOPMENT OF THE CENTER OF THE FACE WITH PARTICULAR REFERENCE TO THE SURGICAL CORRECTION OF BILATERAL CLEFT LIP. R. B. STARK and N. A. EHRMANN, Plast. & Reconst. Surg. 21:177 (March) 1958.

A series of sections were examined from six human embryos with cleft lip and from three normal controls. The problem was to determine which parts belong to the palate, to the lip, or to the columella. Also, does Jacobson's organ belong to the inner nose, or to the columella; and is the philtrum derived from the prolabium, or from the lip processes? The paper is amply illustrated by photographs and drawings of gross material, and with reproductions of microscopical sections. On the basis of this study, the authors propose a new classification of the abnormalities of the lip and palate.

SELTZER, Philadelphia.

THE TREATMENT OF CANCER OF THE FLOOR OF THE MOUTH. J. LEROUX-ROBERT and B. THIBAUT, Ann. oto-laryng. 75:317 (June) 1958.

The authors present 186 cases of cancer of the floor of the mouth. The results are evaluated on a five-year survival basis. The cases that are included in this survey are only those that have not had any previous treatment either by surgery or by radiation.

Treatment depends on two factors—the site of the lesion and its extent. The authors have adopted the following classification: (1) a lesion strictly limited to the floor of the mouth, without involvement of the tongue or invasion of the inferior maxilla; (2) a lesion extending either to the tongue, the gingiva, or the alveolar margin but not involving the bone of the inferior maxilla; (3) a lesion extending to the inferior maxilla and fixed; (4) a lesion of the floor of the mouth extending to the tongue and involving the inferior maxilla.

The following modalities of treatment were used: 1. Radium needles, each containing 0.66 to 1 mg. of radium; 6 to 18 needles were inserted into the lesion, depending on its size and location. These remained in place from 7 to 8 days. 2. A cast was made which contained 4 to 10 tubes of radium, each containing 6.66 or 3.33 mg. of radium. This cast was applied on the surface of the lesion. 3. Roentgen therapy. Here the problem of localization and proper dosage is important. Usually 4,000 to 5,000 r is given over a period of 18 to 20 days in 8 to 10 sessions. Endobuccal and transcutaneous radiation has also been used. 4. Electrosurgery has been used either alone or as a supplement to the above procedures. 5. Supervoltage (1,000,000-2,000,000 volts), cobalt bomb, and betatron radiation have been used in the advanced cases.

In Group I—the most favorable group—there were 19 cases with 9 cures beyond the five-year limit. The modalities used were (*a*) radium needles—six cases with four cures; (*b*) contact—6 cases with 1 cure; (*c*) endobuccal radiation—five cases with three cures. Group II was subdivided into a, b, a plus b, and c. There were 81 cases with 23 cures. All modalities were used

and in certain cases it was necessary to combine several of these. In Group III there were nine cases with no cures. In Group IV there were 13 cases with 2 cures.

Supplemental surgery was necessary in the more advanced cases or where there was an involvement of the regional glands. This was used 8 times in the 34 cases that were reported as cured and 33 times in 88 cases that did not survive.

In summarizing their indications for treatment, the authors have suggested the following routine: for Group I either radium needles implant or endobuccal roentgen therapy. Group II—radium needles; Group III—radium needles plus surgery, and for Group IV surgery. When there is evidence of glandular metastasis—surgery plus irradiation.

PERSKY, Philadelphia.

News and Comment

ANNOUNCEMENTS

Hearing Society Announces Award Competition Opening.—Competition for the 1959 Kenfield Memorial Scholarship, awarded annually by the American Hearing Society to a prospective teacher of lipreading, opened February 1.

Application blanks may be obtained by writing to Miss Ruth Bartlett, chairman of the society's teachers committee, 432 So. Curson Ave., E., Los Angeles 36. Completed applications and recommendations must reach Miss Bartlett by April 1.

Winner of the scholarship will be entitled to take a course in "Methods and Practice in Teaching Lipreading" from any school or university in the United States offering a course acceptable to the teachers committee. A requirement is that the scholarship be used within one year after date of award.

Rules for competition state that a satisfactory applicant shall be a "well-adjusted individual with a pleasing personality, legible lips, good speech and voice, and no unpleasant mannerisms." Graduation from college, with a major in education, psychology, and/or speech, is a requirement.

If the applicant is hard of hearing, he (or she) must have had 30 clock hours of private instruction under an approved teacher of lipreading, or 60 clock hours of lessons in public school classes under a qualified lipreading teacher.

Funds establishing the scholarship were subscribed in 1937 in memory of the late Miss Coralie N. Kenfield, San Francisco, who was widely known for her effective methods of teaching lipreading.

In 1958 the Kenfield award was won by Stuart Edmonds, Midland, Mich.

GENERAL NEWS

Specialists Certified by the American Board of Otolaryngology.—The following candidates were certified as specialists by the American Board of Otolaryngology, Oct. 6-10, 1958:

Arnold, Godfrey E., New York.
Austin, David F., Chicago.
Baldwin, William G., Wenatchee, Wash.
Beatrous, William P., New Orleans.
Becker, Barton R., Pacific Palisades, Calif.
Beekhuis, Gerardus J., Detroit.
Benda, Thomas J., Dubuque, Iowa.
Biber, Jakub J., Delaware, Ohio.
Blanchard, Arthur J., Toledo, Ohio.
Bohcall, Ara, Westbury, N. Y.
Breneman, Fairfax V., Paris, Texas.
Bridwell, Russell E., Overland Park, Kan.
Budoff, Benjamin H., Massapequa, N. Y.
Casey, Dennis A., New Orleans.
Cherken, Harry K., Philadelphia.
Derrick, Cornelius F., Milan, Mich.
Doyle, Samuel C., Hanover, N. H.
Eastwood, Herbert K., Randolph Air Force Base, Texas.
Eves, John H., Waterville, Me.
Farrell, George R., Reno, Nev.
Fidler, Harry E., New Brunswick, N. J.
Fornatto, Elio J., Elmhurst, Ill.
Frame, Stuart M., Rochester, N. Y.
Gladney, John H., St. Louis.
Griffey, Louis E., Natick, Mass.
Gyorkey, Joseph, Chicago.
Haddad, Maurice, Tampa, Fla.
Hamilton, Alfred, Seattle, Wash.
Hanna, Homer H., Phoenixville, Pa.
Iglesias, Robert G., Tampa, Fla.
Kossner, Herbert K., Sacramento, Calif.
Kratz, Robert C., Newport, Ky.
Kwapy, Clemens R., Milwaukee.

Kwok, Francis, New York.
Lange, Rollo DuBois, Madison, Wis.
LeJune, Francis E., Biloxi, Miss.
Lim, Sigdian, Brooklyn.
Lorenz, Elmer W., Takoma Park, Md.
Mackay, Murdo M., Clifton Forge, Va.
Malkoff, Jack, Youngstown, Ohio.
Mandler, George, Chillicothe, Mo.
Mokdessi, George E., Buffalo.
Nito, Yuichi, Memphis, Tenn.
O'Neil, Lloyd F., Peoria, Ill.
Parsons, Richard C., Atlanta.
Pierce, Max K., Los Angeles.
Pinsker, Oscar T., Kansas City, Mo.
Plowman, Edward M., Louisville.
Radpour, Shokri, Brooklyn.
Raggio, Thomas P., Baton Rouge, La.
Razim, Edward A., Berwyn, Ill.
Reasoner, Billy E., Santa Ana, Calif.
Rex, Eugene B., Wynnewood, Pa.
Robinson, Mendell, Providence, R. I.
Santos, Milagros, Rizal, Phillipines.
Simpson, Roger A., Iowa City.
Smersh, Thomas C., Seattle.
Smith, Howard W., New Haven, Conn.
Sofranec, James R., Youngstown, Ohio.
Sophocles, Aris M., Yardville, N. J.
Subba Rao, P. S., Bangalore, India.
Summar, Marion T., Virginia, Minn.
Tucker, Gabriel F., Jr., Baltimore.
Valusek, Fred A., Philadelphia.
Wahl, Milton S., Wilmington, Del.
Walthall, Joseph E., Fullerton, Calif.
Whitaker, Clay W., Cleveland.
Whitfield, Stanley, Peterborough, Ont., Canada
Williams, James A., Sacramento, Calif.
Zavatsky, Albert R., Harrisburg, Pa.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba.

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Eighth International Congress of Bronchoesophagology, Mexico City, March, 1959.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. E. G. Gill, 711 S. Jefferson St., Roanoke, Va.
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Mexican Society of Otolaryngology

President: Dr. Rafael Giorgana
Secretary: Dr. Carlos Valenzuela, Monterrey 47-201, Mexico 7, D. F.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Paul H. Holinger, Chicago.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, Miami, March, 1960.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Victor R. Alfaro, Washington, D. C.
Secretary: Dr. Walter E. Heck, San Francisco.
Meeting: Atlantic City, June 8-12, 1959.

American Academy of Ophthalmology and Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3.
Place: The Homestead, Hot Springs, Va. Time: March 10-11, 1959 (afternoon only).

American Laryngological Association

President: Dr. Fred W. Dixon, 2060 E. 9th St., Cleveland.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Place: The Homestead, Hot Springs, Va. Time: March 8-9, 1959.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: The Homestead, Hot Springs, Va. Time: March 10-12, 1959 (mornings only).

SECTIONS:

Eastern.—Chairman: Dr. Joseph L. Goldman, New York.
Place: The Savoy Plaza, New York. Time: Jan. 8-9, 1959.
Southern.—Chairman: Dr. James T. King, Atlanta, Ga.
Place: Hotel-Atlanta Biltmore, Atlanta, Ga. Time: Jan. 23-24, 1959.
Middle.—Chairman: Dr. Alfred J. Cone, St. Louis, Mo.
Place: Park Plaza Hotel, St. Louis. Time: Jan. 12-13, 1959.
Western.—Chairman: Dr. Robert C. McNaught, San Francisco, Calif.
Place: The Ahwahnee Hotel, Yosemite Valley, Calif. Time: Jan. 17-18, 1959.

American Rhinologic Society

President: Dr. Russell I. Williams, 408 Hynds Bldg., Cheyenne, Wyo.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.

American Otological Society, Inc.

President: Dr. Moses H. Lurie, 483 Beacon St., Boston.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Place: The Homestead, Hot Springs, Va. Time: March 13-14, 1959.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Joseph G. Gilbert, 75 Barberry Lane, Roslyn Heights, N. Y.

American Society of Facial Plastic Surgery

President: Dr. Trent W. Smith, 327 E. State St., Columbus 15, Ohio.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.
Meetings: March 18 and July 15, 1959, New York.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Joseph W. Hampsey, Grant Building, Pittsburgh 19.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6.

Otosclerosis Study Group

President: Dr. E. P. Fowler Jr., 180 Fort Washington Ave., New York 32.
Secretary-Treasurer: Dr. Arthur L. Juers, 1018 Brown Building, Louisville 2.

Society of Military Otolaryngologists

President: Lt. Col. Stanley H. Bear (MC), USAF, Maxwell AFB, Ala.
Secretary-Treasurer: Capt. Maurice Schiff (MC), U. S. N., U. S. Naval Hospital, Oakland, Calif.

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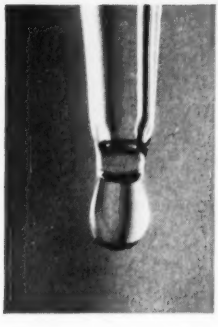

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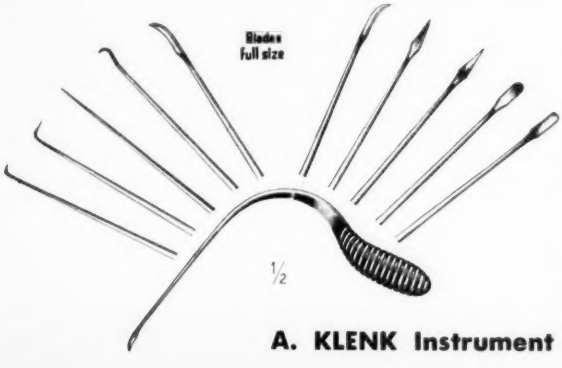

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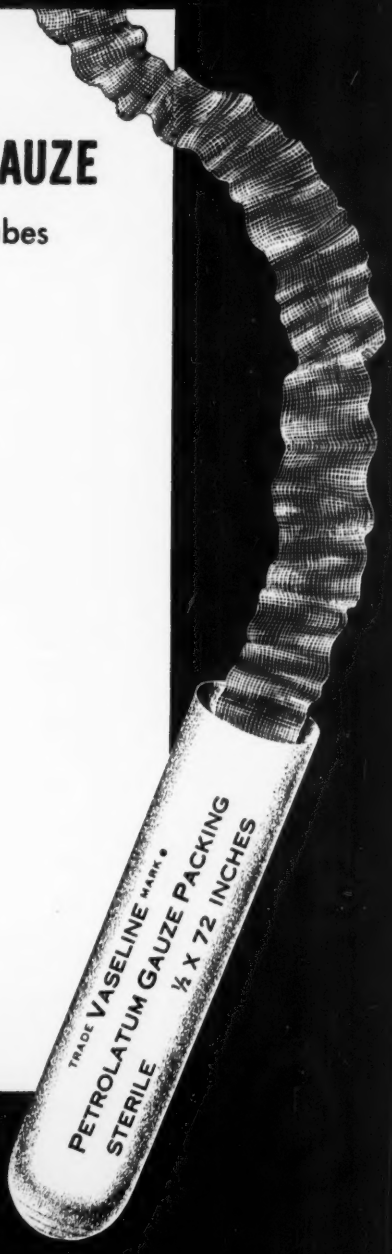

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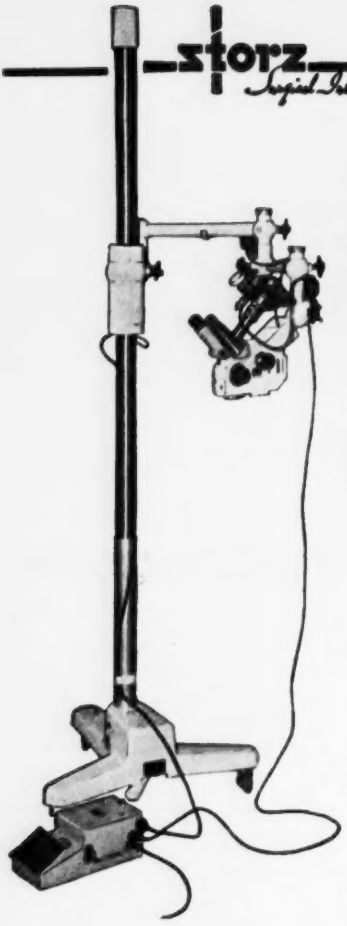
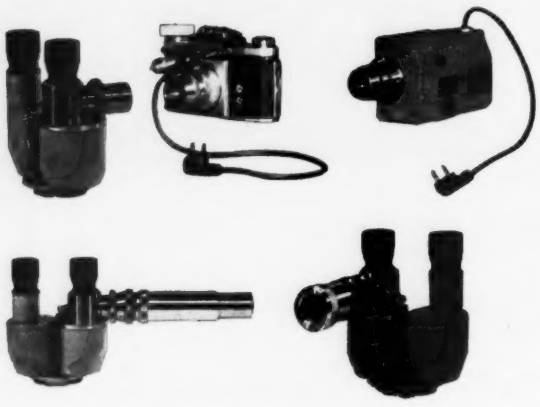


With this equipment it is possible to photograph while operating, without disturbing the operator's vision. Either 35mm slides or 16mm motion pictures may be made by simply interchanging cameras on the binocular take-off tube. This take-off tube may also be utilized to use the auxiliary eyepiece.



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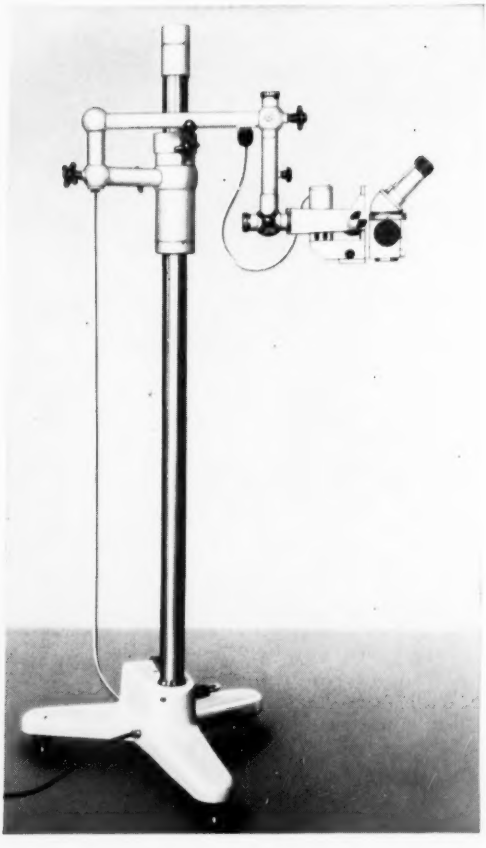

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